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THE FATE OF PRESERVED HETEROGENEOUS GRAFTS OF FASCIA WHEN TRANSPLANTED INTO LIVING HUMAN TISSUES

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THE importance and significance of the use of heterogeneous preserved fascia, especially in plastic surgery, have commanded the attention of many investigators. This present study is concerned with the changes which take place when preserved ox fascia is placed in contact with living human tissue. Such grafts have been studied over periods up to 4 years.

As soon as the animal was killed, the deep fascia was removed, aseptic precautions being used, and the grafts were placed in a solution of 70 per cent ethyl alcohol. While immersed in the alcohol the fascia was cleaned and cut into 2 inch by 2.5 inch pieces and finally transferred into absolute alcohol. After 2 weeks the pieces were considered available for use.

The prepared pieces of fascia were implanted into human tissue during various operative procedures, especially operations for hernia as advocated by Harrison. The implants were removed at subsequent reoperations at intervals up to 4 years in duration.

Specimen 1 is a section of normal preserved ox fascia before implantation into human tissue. Well preserved fibrous tissue is evident. Note that the nuclei are readily seen. The cell outlines are clear, and the cells are arranged uniformly. Blood vessels are not visible. The compact nature of the fascia is evident.

From the Mission Hospital, Bahrein, Persian Gulf.

Specimen 2 is a section of preserved ox fascia 1 week after implantation into human tissue. Very little change is noted. A few leucocytes are seen infiltrating the periphery. No foreign body reaction is seen. The fascial structure is preserved.

Specimen 3 is a section of preserved ox fascia 2 weeks after implantation into human tissue. No marked change is seen in the ox fascia, except that more leucocytes are present at the periphery and fibroblasts are beginning to infiltrate into the graft.

Specimen 4 is a section of preserved ox fascia 3 months after implantation into human tissue. The well defined cell outlines and staining characteristics have changed. There is evidence of absorption at the periphery. Fibroblasts are observed growing into the graft, and capillary growth is also seen. The uniformity of arrangement of the fascial fibers is being changed to an arrangement in bundles, and more cells are seen between these bundles.

Specimen 5 is a section of preserved ox fascia 8 months after implantation into human tissue. There is observed a progressive change in that more fibroblastic growth into the graft is further defining the bundles seen in specimen 4. There is more absorption by the migrating cells at the periphery. No evidence of a foreign body reaction is seen.

Specimen 6 is a section of preserved ox fascia after 2 years of implantation into human tis-

sue. The fibroblasts of the host have grown into the graft very well. Cell outlines in the original graft are largely lost. Capillary invasion is marked, but the transplanted fascia can still be readily identified. There are more leucocytes and migratory cells at the periphery than in specimen 5.

Specimen 7 is a section of preserved ox fascia after 4 years of implantation in human tissue. Good capillary growth is evident at the periphery. A network of collagen fibers and fibroblasts has grown into the fascial graft completing the division into bundles. The nuclei of the original fascial fibers have disappeared. Note the absorption of the peripheral bundles and their extensive replacement by collagen fibers. The preserved tissue can still be easily distinguished from the living tissue.

OBSERVATIONS

The most evident features are:

1. The preserved ox fascia can be identified after 4 years of implantation in human tissue.

2. There is a very slow change in the character and nature of the preserved fascial fibrous tissue cells.

3. There is an abundant growth of capillaries and fibroblasts from the periphery into the fascial graft and complete absorption of the preserved fascial cells at the periphery.

4. The absorption of the cellular detritus of the preserved fascia is noted.

5. The fibers of the preserved fascia become rearranged into bundles as more fibroblastic and capillary ingrowth takes place.

6. There is relatively little leucocytic infiltration at the periphery of the graft and between the bundles.

7. There is no marked foreign body reaction, and giant cells are absent.

DISCUSSION

The use of dead material as grafts has been based principally on the work of Nageotte and Sencert (13, 14, 15). Nageotte (11) in 1916 formulated a theory of the nature and origin of connective tissue. He believed that albuminoid coagula are first formed from the parenchymal cells, that these coagula should not be regarded as living, and that their origin is similar to the formation of blood plasma. He called this coagulum "the fundamental sub-

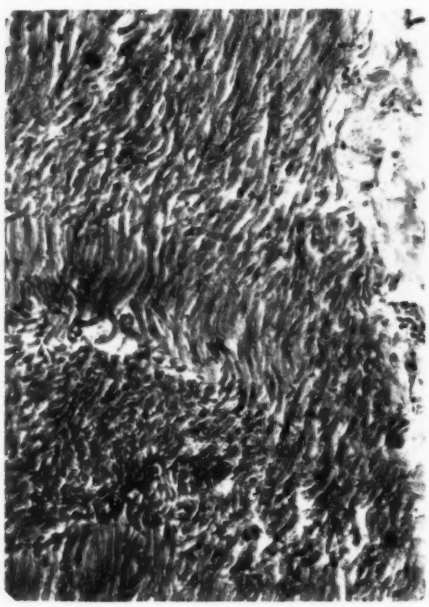

Fig. 1.

Fig. 2.

stance," and he believed that it was composed of elementary collagen fibrils which give rise to collagen fibers, and that connective tissue is formed by the penetration of fibroblasts into the meshes of these fibers. The distinctive feature of Nageotte's theory is that he insists upon the fundamental nonliving character of all connective tissue substances. Baitsell's (2, 3) work supports Nageotte's conclusions. The other theories held regarding the formation of connective tissue are that it is made up of transformed portions of the exoplasm which comes from a syncytium of mesenchyme cells and also that the early syncytium of the mesenchyme cells secretes an amorphous gelatinous nonliving ground substance in which the connective tissue fibers form. Baitsell (4) observed also the direct transformation of fibrin clot into connective tissue fibers. Tissue cells which grew into the fibrin clot developed into fibroblasts.

Nageotte believed that, since connective tissue substances are inert coagula formed from living cells, one would not expect grafts of such tissues to act as foreign bodies and induce phagocytosis, and similarly connective

Fig. 3.

Fig. 4.

Fig. 5.

Fig. 6.

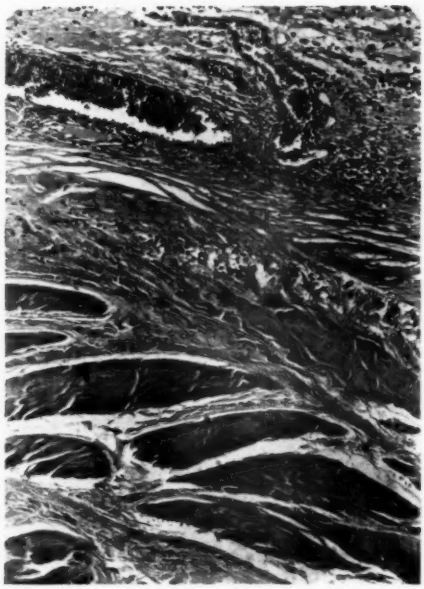

Fig. 7.

tissue preserved in alcohol, since they have the same nonliving origin, should not produce phagocytosis and foreign body reaction. In other words, reaction to preserved fibers should be the same as that to living ones. He explained the results of his experiments on the theory that the nonliving graft takes and becomes adherent; that it soon becomes impossible to determine its limits because the union between the living and nonliving tissue has effected itself so perfectly. In his experimental studies the microscopic examination showed that the implanted graft blended with the living tissue until no line of demarcation could be detected. After the protoplasm of the cells of the preserved graft had been carried off by the migratory cells, or wandering macrophages, new fibroblasts from the host flocked into the persisting connective tissue framework of the graft and established themselves in the place of the old inhabitants. Circulation became established by the growing in of small vessels from the host, and in time it was actually impossible to tell that the graft has been dead when it was implanted. Nageotte called this process the "reviviscence." The only phagocytosis which he was able to observe was that which was necessary for the removal of protoplasm of the preserved graft. Nageotte grafted, with success, cartilage, tendon, segments of arteries, and nerves.

Nageotte (12) and Sencert (17), Auvray, Dustin, Jalifier, Christophe, and many others have grafted successfully preserved animal tendon, nerves, and even an entire patella with good results.

The animal experiments of Koontz are of significance in this connection—heterogeneous and homogeneous grafts did not show any difference—they both healed and did not show any intolerance of the host tissues for the graft. Koontz believed that the preservation of the graft in alcohol eliminates the antagonistic action of foreign protein. It is an important fact that fibroblasts did not grow around the grafts at any time in sufficient numbers to produce encystment. No foreign body reaction was noted by Koontz. He concluded that after a period of a few months it would be impossible to distinguish the living from the preserved tissue graft after that had been transplanted into living connective tissue.

The results of the experimental work of Hass agreed very closely with those of Koontz. He showed that the transplanted preserved fascia united firmly with the muscle and did not tend to take the form of tendinous structures as the live graft usually did. Microscopically close intermingling of the muscle and the transplanted fascia was observed. There was no evidence of a foreign body reaction. There was no difference between the union of preserved fascia with the muscle and that of living fascia. Muscle cells appeared to be transformed into the elements of fibrous tissue and aided in the uniting process.

Rosenblatt and Meyers found very good union between preserved fascia and living tissue although they noted some foreign body reaction around the graft. They believed that the fascial graft acted as an element for substitution of connective tissue and was in part absorbed.

There is no evidence in the observations reported in the present study to support Nageotte's theory of the nature and origin of the connective tissue. From the data obtained in my experiments it is clear that the preserved tissue does not in itself become a living tissue at any time although the value of the preserved tissue is quite evident. When a tissue is dead, its intracellular chemistry changes, and the devitalized material is absorbed and replaced by other tissue cells. The absorption may be slow if the material has been fixed in alcohol. The amount of foreign body reaction depends on the chemical and physical nature of the foreign body; and since fixed connective tissue cells are not irritating, they need not produce such a reaction. However, there occurs sufficient phagocytosis for the absorption of the foreign cellular detritus. This is well seen in the sections.

It may not be advisable to use the expression that preserved tissue unites or heals with living tissue, as used by Nageotte, Koontz, and others. It remains an integral part for a long time but eventually is replaced, at least to a considerable extent, by living fibroblasts to form new connective tissue.

The importance of such preserved fascia lies in the fact that it can be used in various plastic surgery procedures as a framework into which normal tissues may grow. It has sufficient strength to serve in place of normal tissue for long periods of time, and it rapidly becomes firmly attached to these tissues in man.

SUMMARY

A study of the fate of preserved heterogeneous grafts of fascia when transplanted into living human tissue is reported. Such grafts have been observed and studied over a period of 4 years.

The preserved fascia rapidly becomes adherent to the human tissues and remains so for a long period of time. It is gradually replaced by the ingrowth of fibroblasts and capillaries and the formation of human connective tissue. This process was still incomplete, however, after 4 years.

A short review of the experimental studies in fascial transplants is given.

Photomicrographs of sections of preserved ox fascia after periods of from 1 week to 4 years in living human tissue are presented.

The advantage of preserved ox fascia in plastic surgical work is emphasized.

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PERITONITIS OF APPENDICEAL ORIGIN TREATED WITH MASSIVE DOSES OF PENICILLIN

Report of 50 Cases

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THE purpose of this article is to report a group of 50 cases of proved peritonitis of appendiceal origin which were treated with large doses of penicillin over a long period of time. The peritonitis was controlled in all cases and all intra-abdominal masses resolved spontaneously without drainage. The dosage of penicillin was 100,000 units given every 2 hours, intramuscularly, for 2 to 6 days, followed by smaller amounts for 3 to 4 days.

REVIEW OF LITERATURE

In spite of the fact that penicillin has been widely used in the treatment of peritonitis, little has been written on this subject. Craig and others report 7 cases of peritonitis in which 20,000 units of penicillin was given every 2 hours with good results in the majority of the cases. There is no report in the literature of a series of cases of peritonitis treated with large doses of penicillin given over a long period of time. Yet in 1944, Fauley and others reported that in dogs peritonitis induced by ligation of the blood supply of the appendix could be controlled by the administration of penicillin in doses equivalent to 22,500 units per hour given to a 150 pound man over a period of 7 days. The mortality in untreated controls was 92.6 per cent; the mortality in those treated from the time of operation was 0 per cent, and the mortality of those treated after 12 hours was 21 per cent.

It has been established by the work of Ory and others that penicillin given parenterally enters the serous cavities of the body and is present in ascitic fluid in approximately the same concentration as in the blood (7). Loewe and others, moreover, have established that the level of penicillin attained in the blood is almost directly proportional to the dosage of penicillin given intramuscularly or intravenously. When 100,000 units of penicillin is given intramuscularly the level in the blood rises to 1.7 units per cubic centimeter of blood 30 minutes after the injection, falls to 0.8 unit 60 minutes after the injection, and to 0.3 unit after 120 minutes. Craig and others state that a level of 0.02 unit per cubic centimeter is effective for the control of most organisms sensitive to penicillin. It is thus apparent that when 100,000 units of penicillin is given every 2 hours, the lowest levels, occurring just before the next injection, are still over 10 times in excess of minimal effective levels, and the maximum levels are over 80 times as high. There is reason to believe that it is necessary to maintain these high levels over a long period of time if peritonitis secondary to appendicitis or perforation of the colon is to be controlled.

CLINICAL MATERIAL

Definition of peritonitis of appendiceal origin. From November 1, 1944, to November 1, 1945, at the U.S. Naval Hospital, San Diego, over 900 appendectomies were performed. In approximately 70 per cent of these cases, true acute appendicitis was present. In a little over 5 per cent of the cases, peritonitis was found. It is this group of patients, with peritonitis of appendiceal origin, with which this paper is concerned.

For this study no case was accepted as peritonitis of appendiceal origin unless at the time of operation an established purulent peritonitis or appendiceal abscess was present. Cases in which the appendix was ruptured in its removal but in which there was no evidence of an established peritonitis were eliminated.

If operation was deferred and the peritonitis was treated conservatively, such a case was not included unless there was strong clinical evidence of peritonitis subsequently

proved by the development of a palpable mass or by an interval operation confirming the presence of a gross perforation of the appendix. Also excluded from this group were patients who did not develop a mass and who either did not have an interval appendectomy or whose appendix at the time of appendectomy failed to show gross evidence of perforation.

Twenty-five of the 50 patients in this group were considered on clinical grounds to have generalized and 25 to have localized peritonitis. In 8, definite abdominal masses were palpable at the time of admission and in 25, pelvic or abdominal masses were palpable at some time during the course of the illness. All of the patients were sick, and it is clear that their illnesses represent the most serious complications of appendicitis.

The following information concerning the 50 cases of proved peritonitis is of importance: The average temperature on admission was 102.6 degrees, with a high of 105 and a low of 100. The average pulse on admission was 109, with a high of 130 and a low of 90. The average white blood count on admission was 17,000, with a high of 30,000 and a low of 5,000. The average duration of illness prior to entry was 3.2 days. There were 25 patients with general peritonitis, 25 with localized peritonitis, 8 in whom a mass was palpable on admission, 25 in whom there was a palpable mass at some time (15 pelvic and 10 abdominal). Conservative treatment of peritonitis was carried out in 23[1] patients, exploratory operation without appendectomy in 5, immediate appendectomy in 22.

The average duration of the illness before admission of patients subjected to appendectomy was 2.9 days, and of those treated conservatively, 3.8 days. In only 2 cases were the symptoms over a week's duration. Treatment, if any, prior to entry, had consisted in a small percentage of cases, of the administration of sulfonamides and in 2 or 3 instances of small amounts of penicillin. In 5 cases sulfanilamide was placed in the peritoneal cavity at the time of operation, and in 1 case 200,000 units of penicillin solution was left in the abdomen.

A McBurney incision was used in all but 4 of the cases. The stump of the appendix was not inverted, and fine cotton or catgut was used for closure of the abdominal wall.

[1]In all but 3 cases, interval appendectomy confirmed the presence of a gross perforation of the appendix. In the other 3, operation was not performed, but diagnosis was established by the development of a definite mass in the right lower quadrant.

TREATMENT OF PERITONITIS

Penicillin. As soon as the diagnosis of peritonitis was made, all patients were given 100,000 units of penicillin every 2 hours intramuscularly in procaine solution or by continuous intravenous drip. This treatment was continued until signs of improvement appeared. The longest course of this dosage of penicillin was 10 days, the shortest was 1 day, and the average was 3½ days. In the beginning of the period in which these cases appeared, when penicillin was scarce and costly, every effort was made to use as little as possible and the dose was usually halved after the first day. Later it was found more economical to give the larger doses for at least 3 or 4 days and then to continue with 50,000 units every 2 hours or 100,000 units every 4 hours for at least 3 more days. If the large doses were discontinued too soon, recurrences of the peritonitis were apt to appear or the patient developed a localized mass. A second course of penicillin would again result in the subsidence of all symptoms and signs of peritonitis and in the ultimate resolution of the mass (Fig. 1).

In the early days of this experience with penicillin, an attempt was made to set up a standard treatment for all cases. One hundred thousand units was given every 2 hours for the first day, then 50,000 units every 2 hours for 2 days, 50,000 units every 4 hours for 2 days, and finally 25,000 units every 4 hours for 2 days. On several occasions there were recurrences of elevation of temperature and of the symptoms and signs of peritonitis while the patient was still receiving 25,000 units of penicillin every 4 hours. The schedule was changed therefore, and the full dosage was maintained until the clinical signs of peritonitis subsided or until the patient had been on 100,000 units of penicillin every 2 hours for at least 6 days. Four days was sufficient to control the average case, and if satisfactory improvement occurred, the dosage

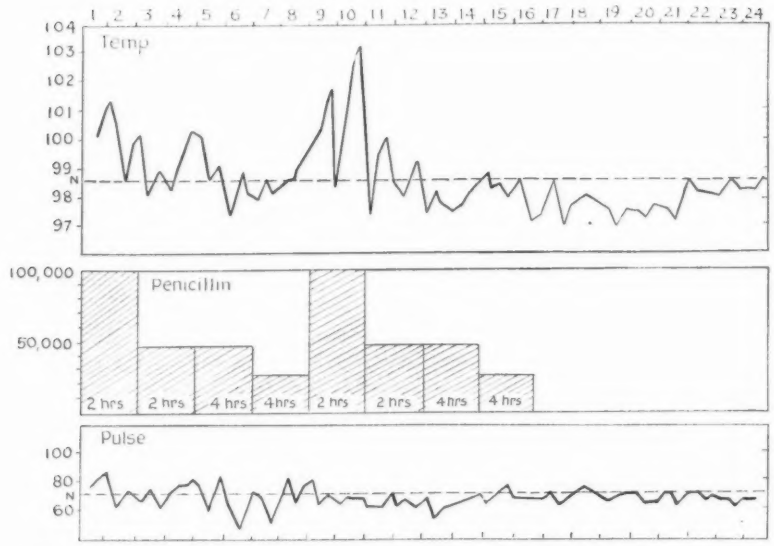

Fig. 1. Localized peritonitis, 2 days' duration, with mass in right lower quadrant. All symptoms and signs subsided on treatment with penicillin. Recurrence when dosage was reduced to 25,000 units every 4 hours. Mass, fever, and symptoms permanently controlled with second course of 100,000 units of penicillin every 2 hours.

was reduced to 100,000 units every 4 hours. Fifty thousand units every 2 hours maintains a better level in the blood, but after 4 or 5 days the patients complain about the injections, and since by this time they are usually feeling much better, it is sometimes difficult to keep their co-operation. It is unwise, however, to discontinue large doses of penicillin on at least a 4 hour schedule before the sixth day of treatment, and sometimes 10 or 12 days may be required before large intra-abdominal masses begin to resolve.

The dosage, 100,000 units every 2 hours, is an arbitrary one and may be higher than required for some, and lower for others. Several patients responded well to 50,000 units every 2 hours, and 1 patient responded slowly to doses of 200,000 units every 2 hours. At least 3 patients receiving 30,000 units every 3 hours showed evidence of the spread of peritonitis or developed recurrences of localized masses with rising temperature and pulse rates when the dosage was lowered to this level. Similarly, several patients who had responded dramatically to large doses of penicillin developed recurrences of their peritonitis when the administration of penicillin was discontinued after 1 or 2 days. In all of these cases the disease was again controlled by the prolonged administration of 100,000 units every 2 hours. Since side effects appear to be no more common when large doses are used than when the usual ones are given, and since the drug is now relatively inexpensive, it would seem safer to err on the side of overdosage. This point of view is supported by experiences in the treatment of sulfonamide or penicillin-resistant gonorrhea, in which studies it has been observed that if inadequate doses are at first given, a resistant strain of gonococcus is apt to develop (3).

There is reason to believe that certain of the commercial preparations of penicillin contain more of the fraction X than others. This fraction is from two to eight times more effective against the hemolytic streptococcus, the gonococcus and other organisms than is the fraction G, although its efficacy against the standard test organism, staphylococcus, is the same (8).

At one time, in this series, there was a group of 14 cases which did not respond as well as the previous cases had, and in checking back it was found that the brand of penicillin had been changed. At the same time, on another service, there was encountered an extraordinarily high percentage of penicillin-resistant cases of gonorrhea. When a different brand of penicillin was used, the penicillin-resistant cases obtained good results. One patient with peritonitis, resistant to the first brand of penicillin, responded promptly to a course of treatment with the second. Five brands of penicillin in all have been used in this series, and all but one have proved satisfactory.

Position. No attempt was made to maintain Fowler's position unless abdominal distention or dyspnea rendered the patient more comfortable in this position. Bed rest was insisted upon until the patient became afebrile.

Diet. Clear fluids were allowed *ad lib.* from the time of entry unless the patient was distended or vomiting. If the patient was unable to take adequate fluids by mouth or if distended, 5 per cent glucose in water or saline was given intravenously to make a total intake of 3,000 cubic centimeters daily.

Gastric suction. Continuous gastric suction was used in patients who were distended or vomiting. The amount of fluid and salt lost by suction was estimated and replaced parenterally. Patients were allowed to drink clear fluid.

Miller-Abbott tube. If gastric suction failed to decompress the abdomen or if the returns from the stomach indicated that ileus was present, a plain plate of the abdomen was taken. If this showed the slightest evidence of small intestinal obstruction, a Miller-Abbott tube was passed without delay. As soon as the Miller-Abbott tube passed the pylorus, the patient was allowed to take clear fluids, and when the tube had entered the ileum a soft diet was given.

It was not found necessary or desirable to use hot stupes on the abdomen. None of the patients in this group was seriously ill more than 4 days, and none went more than 4 days without taking food by mouth. For this reason, the glucose was not supplemented with amino acids. Since the plasma proteins did not fall and since edema occurred in no case, plasma was not necessary. In no instance did anemia below 65 per cent hemoglobin occur, and no transfusions were given. The leucocytes showed little alteration during the febrile part of the illness and rechecked leucocyte counts while the patients were still ill, averaged 16,000 as compared to 17,000 on admission. In no instance did the leucocytes fall below 5,000 and almost without exception immature forms and toxic granulations decreased. One patient entered the hospital 3 days after an appendectomy for a perforated appendix. At the time of admission his temperature was 105, pulse 120, and he had classical signs of a generalized peritonitis. His white blood cells were only 5,000 and the differential blood count showed a marked leucemoid reaction, with 6 myeloblasts, 2 premyelocytes, 14 myelocytes, 11 juveniles, 13 band forms, 33 segmented forms, 11 lymphocytes, 8 questionable myeloblasts, and 2 unclassified cells. The leucocyte count rose steadily and the leucemoid reaction subsided following the administration of large doses of penicillin.

Oxygen was not used and distention was controlled, when necessary, by gastrointestinal intubation.

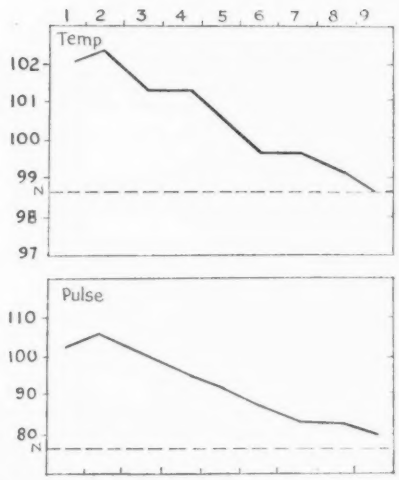

Fig. 2. Composite temperature chart of 21 consecutive cases of peritonitis treated with large doses of penicillin.

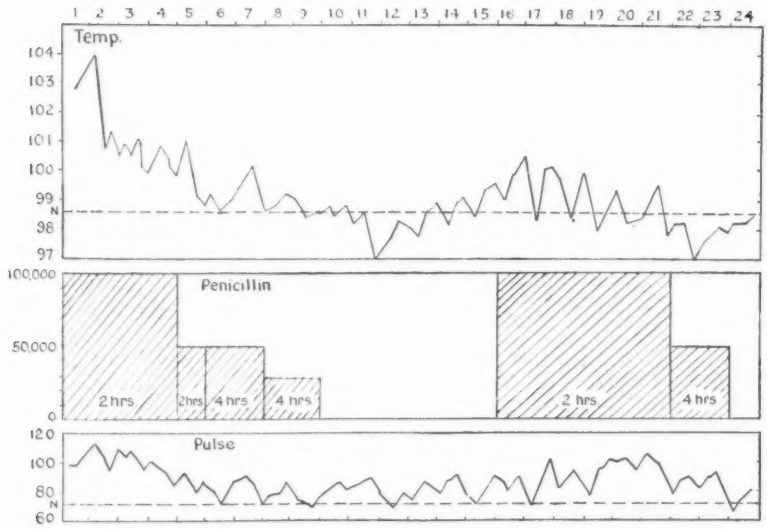

Fig. 3. Appendicitis of 2 days' duration. Appendectomy with finding of diffuse peritonitis. Peritonitis controlled with large doses of pericillin. Five days after penicillin was discontinued symptoms recurred and large mass, palpable by both rectal and abdominal examination, filled the pelvis. Second course of penicillin resulted in resolution of mass.

COURSE OF PERITONITIS

The course of the peritonitis in these 50 cases differed in several striking respects from that seen prior to the use of penicillin. The patients did not appear sick after the first 2 days. In no instance did the peritonitis appear to spread after the first 24 hours of treatment, and in all cases but one, definite clinical improvement had occurred by the fourth day. The abdomen lost its exquisite tenderness after the second day, and rigidity simultaneously disappeared. Distention was not often seen and ileus was minimized. Many of these patients did not vomit and were able to take a full liquid diet throughout the course of the disease. Most of them were alert, read magazines or newspapers, and did not seem to be uncomfortable.

Temperature. In no instance did the temperature or pulse rate rise higher after the first 2 days of treatment. In 50 per cent of the cases the temperature was normal by the end of the fifth day of treatment. The average febrile period for the entire group was 8 days before the temperature failed to rise over 99 for a 24 hour period. Nine patients ran a low-grade fever, fluctuating from normal to 100 for over 10 days, and 1 patient remained febrile for 21 days. In the latter case a drain had been left in the pelvis and there was a profuse purulent drainage and wound infection, but the temperature did not rise over 100. A composite temperature chart of the first 21 cases treated with large doses of penicillin is depicted in Figure 2. The temperatures of the last 29 cases reached normal in exactly the same number of days as the first group, but their charts were not available for incorporation in the graph.

Pulse rate. One of the characteristic signs of uncontrolled peritonitis is an elevation of the pulse rate out of proportion to that of the temperature. Yet in this group the highest recorded pulse rate of the day had fallen below 100 by the third day (average) and in only 4 instances did the pulse rate remain over 100 longer than 1 week.

Ileus. Most patients with peritonitis experience considerable ileus, become distended, vomit, or put out large amounts of green or brown material by gastric suction. The majority in this group did not show signs of pro-

nounced ileus, or if they did so, it was only for a day or two. One explanation of this phenomenon is that penicillin controls the invasive qualities of the bacteria and minimizes the tendency to produce a paralytic type of ileus. This absence of cellulitis likewise may explain the rapidity with which the tenderness and rigidity of the abdomen subsides. The relatively slight amount of ileus and the consequently minimal loss of fluid and chlorides renders these patients easy to keep in fluid and chloride balance. Few alterations in blood chlorides were observed.

Despite the low incidence of paralytic ileus, true mechanical obstruction of the small bowel was fairly common. In 13 cases there was x-ray and clinical evidence of obstruction and in 12 of these 13 cases the obstruction was associated with the presence of a palpable mass. It is thus probable that the ileus was the result of mechanical obstruction in an inflammatory mass rather than secondary to the paralytic action of a generalized peritonitis. In every case the passage of a Miller-Abbott tube corrected the obstruction and in only 1 instance did it recur and require a second decompression.

Masses. During the acute stage of peritoneal irritation there is often so much rigidity that it is impossible to determine whether or not a mass is present. In only 8 cases was a definite mass palpable at the time the patient entered the hospital. But at one time or another during the course of the disease, 50 per cent of the patients developed intra-abdominal masses, 15 patients developed a pelvic mass palpable only by rectum and 10 developed masses easily palpable through the abdominal wall. All of these masses were located in the lower abdomen.

It has been estimated by Ochsner and Johnston that in cases of appendiceal peritonitis treated conservatively, three-fourths will subside spontaneously without going on to abscess formation (6). These authors state that in the remaining 25 per cent, incision and drainage of the abscess will be necessary. In this series of 50 patients, 26 were treated without appendectomy or drainage of an appendiceal abscess, 23 of these having had no operation and 3 having been merely explored.

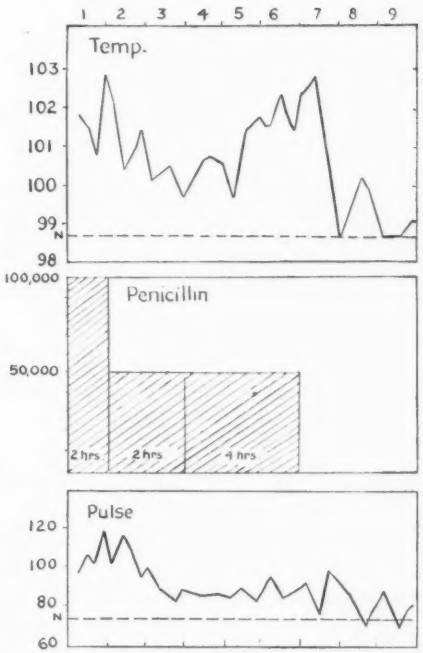

Fig. 4. Secondary rise of temperature due to penicillin. Slow pulse and prompt return to normal of temperature curve occurred as soon as administration of penicillin was discontinued.

In 15 patients a mass developed, but under treatment with 100,000 units of penicillin every 2 hours, 4 to 8 days, they became afebrile and free of symptoms; the masses lost their tenderness, became smaller, and eventually disappeared. In no instance were incision and drainage necessary and insofar as it was able to be determined, there was no instance in which pus was discharged spontaneously into the bowel. In several patients, symptoms recurred after being once controlled, and the mass became larger after treatment with penicillin was discontinued, but in each a second course of treatment resulted in a second remission and eventually in complete resolution of the mass. Figure 3 depicts the temperature chart of a patient who required a second course of penicillin to control a pelvic mass.

Some of the masses were so large as to cause a visible asymmetrical deformity of the abdomen or to fill the pelvis so completely that on rectal examination the rectum appeared to

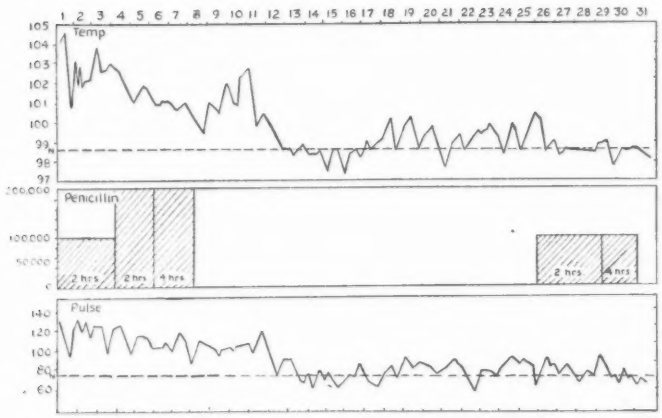

Fig. 5. Localized peritonitis with mass in right lower quadrant and pelvis. No operation. Patient apparently refractory to second course.

be occluded. In 2 instances fluctuation could be detected by rectal examination, and attempts were made to provide drainage by forcing a passage through the rectal wall. Unlike the usual pelvic abscess when such a passage is accomplished with ease, it was found that the tissues were not friable and that the wall of the rectum was so normal as to render difficult the passage of finger or instrument. The attempt was therefore abandoned, and the fluctuant area gradually disappeared and the mass resolved. Passage of pus was never detected.

Another remarkable finding about the masses is the absence of acute tenderness. They are sensitive to deep pressure, but when they are resolving they present few of the features of an appendiceal abscess. In the case of one man in his fifties, the absence of tenderness and the presence of a hard fixed mass in the right lower quadrant led to the suspicion that the peritonitis had originated from a carcinoma of the cecum. Subsequently, it was proved that the appendix was perforated.

In 7 cases, intra-abdominal masses developed following appendectomy. Rogers and Faxon encountered 40 such cases at the Massachusetts General Hospital. In 50 per cent of these patients the masses resolved spontaneously, 16 required abdominal drainage, and 4 required rectal puncture. Four patients died. None of the 7 patients in this series required drainage and all resolved spontaneously. In 1 or 2 instances other than these 7 cases there may have been spontaneous drainage of small amounts of pus from the abdominal cavity through the wound but it was not possible to tell whether the abscess was deep in the abdominal wall or superficially located in the peritoneal cavity. In any case, all definite intra-abdominal masses resolved spontaneously without surgical intervention.

Infection of wounds. The administration of penicillin may cause a slight reduction in the incidence of wound infection, but it will not prevent it. Thirty-six per cent of the wounds became infected, but in no instance did a severe infection develop. The remarkable thing about the wound infections was the absence of severe local or systemic reaction. Often they behaved like cold abscesses, pointed, became fluctuant, and discharged spontaneously without making themselves evident by pain, a significant febrile reaction, or redness or tenderness of the tissues surrounding the wound. In no instance was there extensive cellulitis. Provided no deep sinuses were present, the wounds healed with remarkable rapidity after the discharge of the pus.

Side effects of penicillin. Approximately 100 patients treated with 100,000 units of peni-

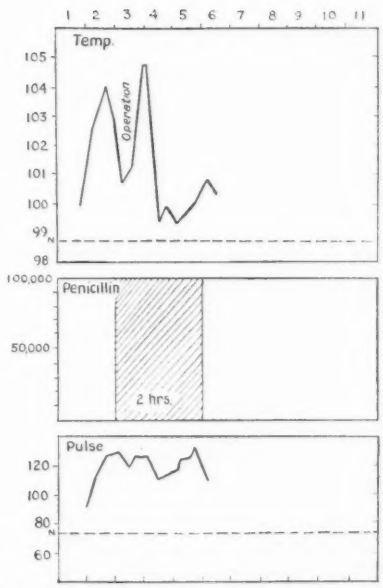

Fig. 6. Diffuse pelvic peritonitis due to perforation in carcinoma of sigmoid. Penicillin instilled in peritoneal cavity and given parenterally. Death by pulmonary embolism on third postoperative day. Autopsy showed no peritonitis.

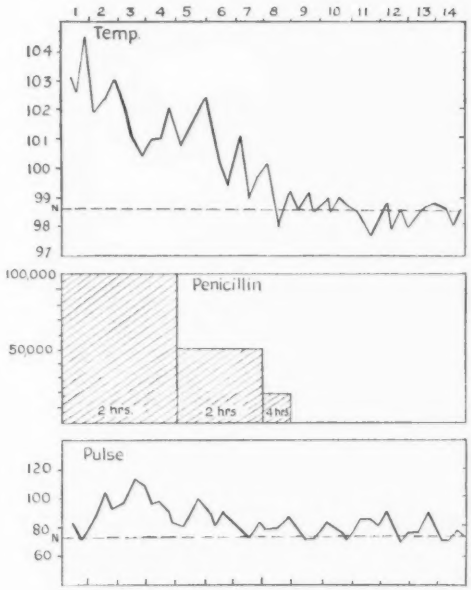

Fig. 7. Severe peritonitis. No operation.

cillin every 2 hours for varying lengths of time had no serious side effects. Urticaria is the most distressing symptom, but it is rare and seems to occur no more frequently with large than with small doses.

Fever may occur and it is characterized by elevation of the temperature without a corresponding elevation of the pulse rate or increase in the clinical manifestations of the disease (Fig. 4).

SUMMARY OF CLINICAL COURSE

The clinical course of peritonitis of appendiceal origin treated by large doses of penicillin given over a long period of time can be summarized as follows:

1st and 2nd day. No apparent change in the general condition of the patient. Peritonitis does not appear to spread.

3rd day. Improvement in patient's clinical condition; pulse and temperature are lower; patient feels better; ileus subsiding; abdomen less rigid and tender.

4th day. If the patient is not definitely improved it is likely that mechanical small intestinal obstruction is present. Roentgenogram of the abdomen should be made.

5th and 6th day. If there is no obstruction, patient is usually feeling well, eating a soft diet, and is alert and comfortable.

7th day. Occasionally intra-abdominal masses persist, but there is little fever and no symptoms.

8th to 12th day. If penicillin has been discontinued, signs of recrudescence of infection in abdominal abscesses should be looked for. A low-grade fever may be disregarded, but if it rises higher daily, or is accompanied by increasing pain, tenderness, or "gas pains," a second course of penicillin should be given.

Although this summary characterizes the course of the average case, an occasional resistant case will be encountered. If this occurs, one should not be discouraged but should continue treatment for 8 to 10 days. Eventually a response is usually obtained (Fig. 5).

Results of treatment of peritonitis with penicillin. In all of the 50 cases of proved peritonitis of appendiceal origin reported in this series the peritonitis has been controlled by the administration of penicillin. No intra-abdominal abscesses have drained spontaneously into the bowel or have required surgical

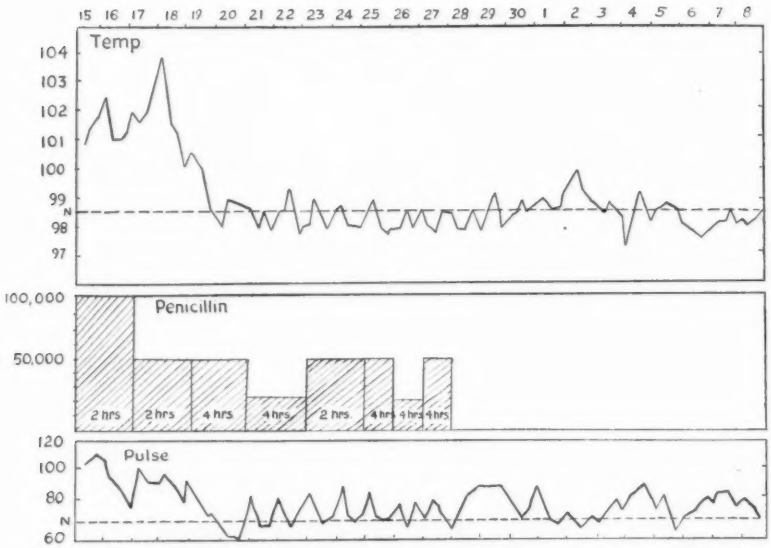

Fig. 8. Diffuse peritonitis. No operation. Large pelvic mass gradually absorbed without discharge of pus.

drainage. All intra-abdominal masses have eventually resolved, although several have required two or three courses of treatment. There have been no deaths as a direct result of peritonitis. The 1 death that occurred was due to thrombosis of the mesenteric veins, an indirect result of peritonitis which at the time of death was well controlled.

CASE REPORT

The patient was a young man who entered the hospital with a history of abdominal pain of 12 hours' duration. Abdomen was rigid enough to suggest the diagnosis of a perforated ulcer. Operation was performed immediately and what appeared to be a generalized purulent peritonitis was found. A culture showed Bacillus proteus and Escherichia coli. The appendix was perforated near its base.

Convalescence was uneventful for 3 days. The temperature fluctuated between 100 and 102, and the pulse rate remained elevated. On the third postoperative day the patient appeared a little better and the abdomen was somewhat softer. Later in the day his pulse became rapid and within a few hours it was imperceptible. The patient became delirious, then comatose, and despite supportive therapy, expired.

Postmortem examination revealed the general peritoneal cavity to be clean and glistening. An abscess approximately 1 inch in diameter containing thick white pus was present in the right iliac fossa and a similar one was in the left. In the pelvis was another encapsulated abscess about 2 inches in diameter. The remainder of the peritoneal cavity was filled with an enormous amount of clear, blood-stained fluid, and the lumen of the small bowel was filled with this same material. Further examination revealed a diffuse thrombosis of the smaller mesenteric vessels. The remainder of the examination showed nothing remarkable.

This patient unquestionably died as the indirect result of peritonitis, but the localized and completely walled-off abscesses were not sufficient to have caused his death had he not developed a thrombosis of the mesenteric veins. This was the only fatality from appendicitis among the 900 patients operated upon in the past year.

Peritonitis secondary to perforating lesions of the large bowel appears to respond as readily to penicillin as does that due to appendicitis, provided of course that the contamination of the peritoneal cavity is controlled by exteriorizing or resecting the involved segment. Three patients with perforated carcinomas of the colon and one with a perforated diverticulitis of the colon have been operated upon and the involved bowel resected. In all cases from 12 to 36 hours had elapsed since the perforation and a diffuse purulent peritonitis was present. Yet in all cases the peritonitis was controlled. One patient expired as the result of a massive pulmonary embolism on the third postoperative day. A carcinoma of the sigmoid had perforated nearly 36 hours before. The pelvis contained several hundred centimeters of thick pus and the entire abdomen was rigid and gave

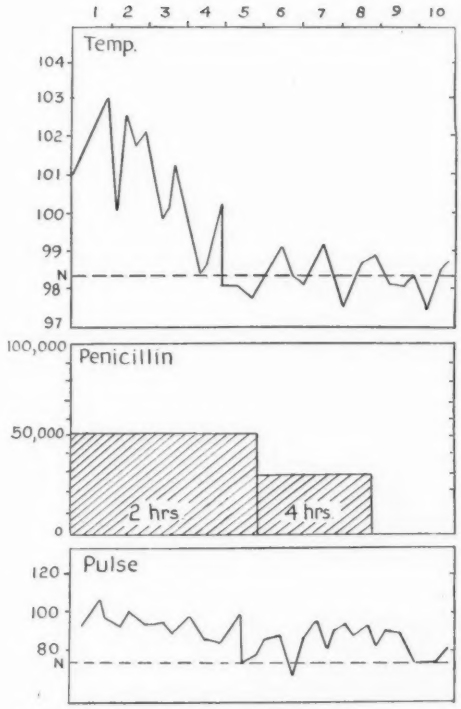

Fig. 9. Mass in right lower quadrant. No operation.

the appearance of a generalized peritonitis. The carcinoma was resected, the pus was aspirated from the pelvis, and 200,000 units of penicillin was placed in the peritoneal cavity. Two hundred thousand units was given intravenously and 100,000 units every 2 hours. On the third postoperative day, the temperature was between 99 and 100.5, the pulse between 100 and 130, and the abdomen was soft, flat, and not tender. The patient expired suddenly. Autopsy showed a massive pulmonary embolism, but aside from a little redness of the peritoneum, there was no evidence of peritonitis and no pus or fluid was present (Fig. 6).

In this series, the results of the management of appendiceal peritonitis have been much the same whether immediate operation was performed or whether the peritonitis was managed conservatively. In each case the average length of time before the temperature fell to normal was 8 days. Masses were palpable more frequently in the patients whose appendices were not removed. Mechanical intestinal obstruction, associated with the masses, occurred more frequently in the group not operated upon.

In the group operated upon, on the other hand, the patients appeared somewhat sicker, and experienced a more profound toxic reaction from the peritonitis.

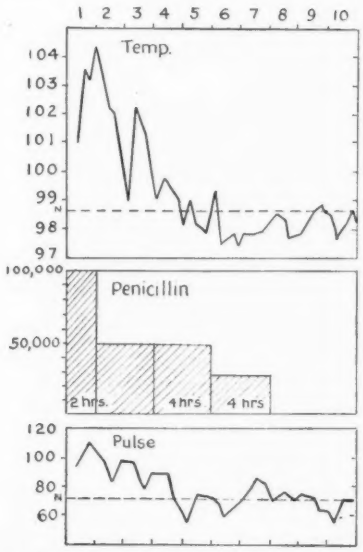

Fig. 10. Abdomen explored. Abscess found but not disturbed. Appendix not removed.

The only death that occurred was in the operated upon group. Since intestinal obstruction of inflammatory origin is easily corrected by decompression with a Miller-Abbott tube, and since little can be done to combat the overwhelming toxemia incident to operation performed in the face of a fulminating peritonitis, it is my impression that conservative treatment is the safer method of dealing with seriously ill patients. In most cases, and especially when the general condition of the patient is good and sufficient penicillin is given to control the peritonitis, there is little to choose between the two methods of treatment (Figs. 7 to 12).

EVALUATION OF STUDY

There is strong evidence that peritonitis of appendiceal origin in both man and dog can be controlled by large doses of penicillin given over a long period of time. There is also evidence that penicillin levels of the blood are proportional to the amounts given intramuscularly and that the drug is present in ascitic fluid in about the same concentration as in the blood. Yet penicillin in concentration up to 20 units per centimeter does not inhibit the growth of Escherichia coli (1). Escherichia coli and other penicillin resistant organisms are almost always found in the cultures taken from patients with appendiceal peritonitis.

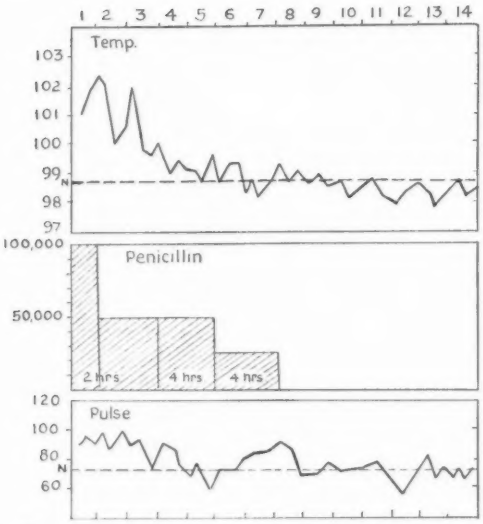

Fig. 11. Appendiceal abscess drained but appendix not removed.

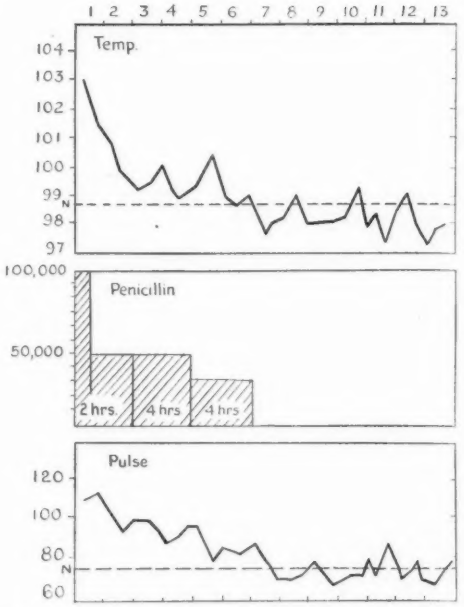

Fig. 12. Appendiceal abscess ruptured at operation. Appendix removed.

How can it be explained that peritonitis due to mixed infections is controlled by concentrations of penicillin less than 1/10 of the minimum level required to inhibit the growth of Escherichia coli, the predominant organism in the infection? And why is it that whereas in most infections the results of chemotherapy are prompt and dramatic, manifesting themselves within 24 hours of the initiation of treatment, in the treatment of peritonitis the results become apparent slowly? And, finally, why is it that four or five times the usual effective dose of penicillin is required to obtain a satisfactory result?

These questions can best be answered in the light of Altemeier's studies. Altemeier has shown that cultures of Escherichia coli or of Bacillus pyocyaneus have the property of destroying the activity of penicillin. If a known amount of penicillin is incubated with a culture of Escherichia coli, 98 per cent of the activity of the penicillin is destroyed in 12 hours.

This ability of Escherichia coli to destroy the activity of penicillin could well account for the fact that large doses of penicillin are required to control the gram positive cocci when they are growing in a mixed infection and are associated with Escherichia coli. It also explains why it takes several days and large amounts of penicillin to control infections due to a mixture of Escherichia coli and gram positive cocci. By giving enough penicillin over a long period of time eventually more penicillin is given than the Escherichia coli can destroy, and finally there is built up a sufficient concentration of active penicillin to inhibit the growth of the virulent cocci.

Although it can now be seen how it is possible for large doses of penicillin given over a long period of time to inhibit the gram positive cocci, there is still no explanation as to why penicillin, in concentrations insufficient to inhibit Escherichia coli, eventually results in control of infections in which this organism predominates. It is possible that Escherichia coli, in pure culture, is not a pathogenic organism, but is in reality a mere saprophyte. If this were the case one need not rely on chemotherapy to control it, as the normal defense mechanisms of the body would be able to deal with it once the gram positive cocci were inhibited. The absence of cellulitis around the

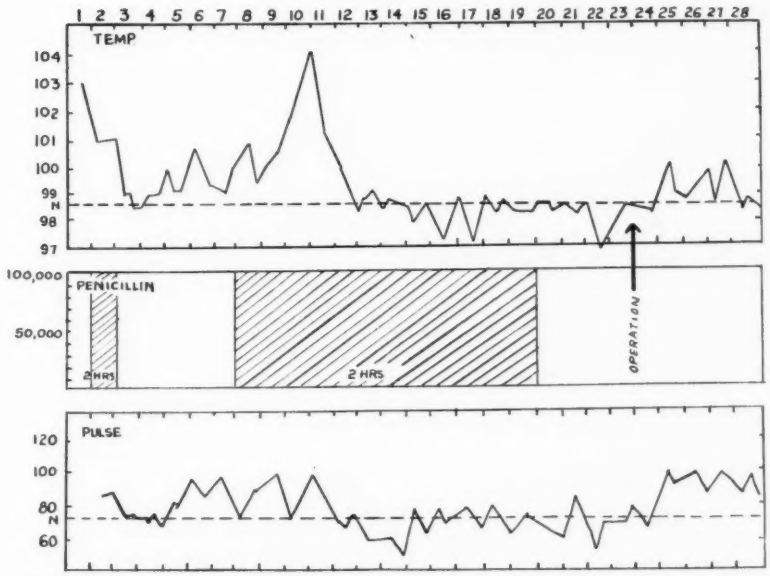

Fig. 13. Acute appendicitis. Patient received 100,000 units of penicillin every 2 hours for 1 day. Penicillin then discontinued and within 5 days there was recurrence of signs and symptoms of localized peritonitis. Second course of penicillin then given over a period of 12 days. Temperature again fell to normal and signs of peritonitis disappeared. Ten days after patient had become afebrile, operation was performed and abscess containing several ounces of foul pus with Escherichia coli odor was found. Since systemic reaction had subsided and the mass was steadily decreasing in size, it is probable that this mass would have been absorbed spontaneously without surgical drainage.

infected wounds of patients receiving large doses of penicillin, the absence of tenderness of large intra-abdominal masses, the absence of friability of the tissues overlying these masses, the absence of paralytic ileus in the face of diffuse peritonitis when adequate doses of penicillin have been given for a few days, all of these findings and the frequent observation that the temperature may be normal and the patient may feel well in the presence of a large intra-abdominal mass, point to the fact that it is not Escherichia coli but the gram positive cocci that are the chief offenders in peritonitis. If the cocci are controlled, the peritonitis is controlled and the marvelous defensive and absorptive mechanism of the peritoneum is able to take care of the saprophytic or weakly pathogenic Escherichia coli infection.

In wounds, the defensive and absorptive action of the peritoneum is not available and, moreover, there is suture material, strangulated tissue, serum, and blood, in which Escherichia coli can grow. Even if it does not invade and cause a cellulitis, a wound infection of low virulence is apt to develop. Instead of being absorbed, as it might be in the peritoneal cavity, eventually it drains. This explains the persistence of a normal expectancy of wound infections despite treatment with penicillin.

In only 1 case in this series was an inflammatory mass opened while it was still palpable. This patient had had 100,000 units of penicillin for 6 days and had been afebrile for 11 days. He had no symptoms, but a mass was still palpable. An interval appendectomy was advised and at operation, 4 or 5 ounces of foul, fecal-smelling pus was obtained and the general peritoneal cavity was extensively contaminated. Yet there was little systemic reaction and the temperature did not rise over 100. Unfortunately, no culture was taken, but despite the fact that Escherichia coli must once have been present to produce

Units of penicillin per c.c. of medium.	10	5	2.5	1.25	0.625	0.312	0.156	0.078	0.039	0.019	0.0097	0
Pure culture Staphylococcus aureus	–	–	–	–	–	–	–	–	–	+++	+++	++++
Mixed culture Staphylococcus aureus	–	–	–	–	–	–	+	+	++	++	+++	+++
Mixed culture Escherichia coli	+++	+++	+++	+++	+++	+++	+++	+++	++	++	+	+

the odor, the patient had no systemic reaction and the mass was becoming smaller each day and probably would have resolved spontaneously (Fig. 13). If sufficient penicillin is given to destroy the gram positive cocci, it would appear that the peritoneum can take care of the rest.

SUMMARY AND CONCLUSIONS

1. A group of 50 patients with proved appendiceal peritonitis treated by the administration of 100,000 units of penicillin every 2 hours for several days is reported.

2. There was but 1 death in this series and this did not occur as the direct result of peritonitis but from thrombosis of the mesenteric vessels.

3. In all cases of spreading peritonitis, the infection was controlled by penicillin.

4. In all cases of localized peritonitis with or without formation of a mass, the infection was controlled and the mass eventually was absorbed.

5. In no case was it necessary to drain an intra-abdominal abscess and so far as is known, there was no spontaneous drainage into the bowel.

6. It is suggested that the mechanism of action of large doses of penicillin in mixed infections is against the virulent gram positive cocci. Escherichia coli is known to inactivate penicillin, hence large doses of pencillin must be given for long periods of time if effective action against the cocci is to be obtained.

7. It is further suggested that Escherichia coli in pure culture usually is of low virulence and that if the virulent gram positive cocci are controlled by chemotherapy, the peritoneum is able to localize and eventually absorb infections due to the remaining Escherichia coli.

NOTE.—To check the in vitro resistance of mixed cultures to penicillin, a heavily seeded 18 hour agar slant of staphylococci Strain N.R. RL 209 was washed with a 6 hour broth culture of Escherichia coli. This was diluted to make a faintly turbid suspension and 0.01 cubic centimeter of this was used as the inoculum.

After 24 hours' incubation at 37 degrees smears were made of all tubes and stained by Gram's method (see table).

It is apparent that concentrations of penicillin of 0.030 unit per cubic centimeter inhibited the staphylococcus in pure culture, but that concentrations of 0.312 unit per cubic centimeter were required to inhibit the staphylococcus in the presence of Escherichia coli. The bacteriology was conducted by Mr. Alfred Reich of the Cleveland Clinic.

The above figures indicate that approximately 8 times as much penicillin is required to control staphylococci in mixed culture without Escherichia coli as when the staphylococcus is in pure culture. This figure corresponds quite accurately with the increased dosage required to control mixed infections as compared with infections caused by gram positive cocci in pure culture.

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TORSION OF THE SPERMATIC CORD

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TORSION of the spermatic cord has been discussed and reported in the medical literature since 1840. Delasiauve described the first clinic case. In May, 1944, Ewert and Hoffman stated that the literature including June, 1943, revealed 489 cases of spermatic cord torsion. Many cases of torsion diagnosed at operation are never reported. Many cases in which operation is not done remain undiagnosed and the result is aseptic necrosis and late atrophy. The literature indicates that this urological emergency is still not recognized sufficiently early to save many of these testes by surgical intervention. In 80 reported cases 64 (80 per cent) had been operated upon and in only 15 (23.5 per cent) could the testis be saved by detorsion. In 49, or 76.5 per cent, of the cases orchiectomy had to be performed for testicular aseptic necrosis. The remaining 16 (20 per cent) cases showed that 5 had spontaneously reduced themselves; in 3 the detorsion was accomplished by the patient; in 5 immediate surgery was refused and later follow-up showed complete atrophy; in 3 others the diagnosis of torsion can well be questioned.

ETIOLOGY

A great deal has been written about the etiology of torsion, but some factors still remain obscure. In the absence of trauma there exists a congenital anomaly which predisposes to acute or recurrent torsion. Torsion of the spermatic chord may occur outside of the tunica vaginalis (extravaginal) or inside of the tunica vaginalis (intravaginal). Extravaginal torsion occurs more readily in undescended testes in the presence of trauma and is much less common. Lowsley and others deny the occurrence of extravaginal torsion and accept only the case reported by Taylor in 1897. The intravaginal type of torsion is most common and may occur in undescended and descended testes alike. Existing anomalies predisposing to intravaginal torsion are: imperfect descent, absent or long mesorchium, absent or long gubernaculum, high investment of the tunica vaginalis, intravaginal insertion of cremasteric muscle, and redundant scrotum. In addition, the vascular pedicle does not enter the testis through the body but rather through the upper pole. With such an anomalous "cherry stem" vascular pedicle, an absent mesorchium and high investment of the tunica, we have a perfect anlage for intravaginal torsion of the spermatic cord. Muschat has demonstrated cremasteric fibers inside of the tunica vaginalis which he believes under the right conditions are responsible for torsion. Indirect muscular exertion and trauma frequently precede torsion. There are other cases, however, such as those occurring during sleep, in which the exciting factor still remains obscure. The scrotal structure which precipitates torsion is the contraction or segmental contraction of the cremasteric muscle.

This condition may occur at any age; Hegner and Postma and Langley reported inguinal torsion in 4 months old infants and O'Conor reported one in a man 68. The majority occur before the 20th year. O'Conor in 124 cases found the average 13.3 years, and Abeshouse in 350 cases the average age 17.7 years, Clute states that 47 per cent occur between the ages of 15 and 25. Ewert and Abeshouse report that the right and left sides are equally involved. Twenty-six cases of bilateral spermatic cord torsions have been recorded. Scudder reported that about one-half (47 per cent) occur in undescended testes.

SYMPTOMS

The history of acute excruciating pain in the testis of a patient who has unassociated urinary tract infection should lead us to suspect an acute torsion of the cord. While this

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acute pain may follow any type of muscular effort or exertion, it can awaken a patient out of a sound sleep. Patients may become nauseated, vomit, and observe rather soon a sudden swelling of the involved testicle. The pain may be most severe during the first 24 hours and after 48 to 72 hours it will spontaneously begin to subside. When the testis is in the groin or within the abdomen the corresponding thigh may be held in the semiflexed position. In the presence of urinary tract infection or gonorrhea, the diagnosis of acute torsion becomes exceedingly difficult.

Before the age of 20, a history of recurrent pain in one or both testes, in the absence of infection, should lead us to suspect chronic recurrent torsion in every patient.

PHYSICAL FINDINGS

The physical findings in the acute and chronic cases are quite dissimilar. In acute cases there is an early swelling, edema, and tenderness involving the affected scrotal sac. Prehn in 1937 noted in acute epididymitis that elevation of the testis would tend to relieve the pain while in acute torsion the pain was always aggravated (Prehn's sign.) Also Ownby and Atkinson in 1942 emphasized the early edema and fixation of the scrotal contents to the adjacent coverings. They stated that when present Dr. James Dillon had repeatedly called their attention to this valuable diagnostic aid (Dillon's sign.) Often the testicle will be elevated but due to the edema it is impossible to differentiate the testicle from the epididymis. The cord above the testicle is usually enlarged. In the absence of infection there are usually no additional urologic abnormal physical findings. The white blood count may be elevated even as high as 20,000. The pulse and temperature usually are normal. In neglected cases of spermatic cord torsion there may be still some enlargement but the outstanding findings are the rubbery, firm, smooth, testicular consistency with elevation and retraction of the testis against the external inguinal ring (Fig. 1). In late cases the testes become smaller and asymptomatic, finally ending in complete atrophy. The epididymis is usually involved and causes the irregularities noted on palpation.

DIAGNOSIS

The diagnosis in many instances should be suspected from the history. Many patients give a history of recurrent testicular pain and swelling. The findings of acute swelling following sudden pain in the testis and with all other examinations negative, strongly suggest torsion of the spermatic cord. It is usually impossible to differentiate between the epididymis and testicle in acute cases. When present Prehn's and Dillon's signs may be helpful. It is of extreme importance to make an early diagnosis if the testicle is to be saved.

DIFFERENTIAL DIAGNOSIS

Acute epididymitis. Acute epididymitis in many ways may so simulate acute torsion that a differential diagnosis becomes extremely difficult. In the presence of urethral discharge, pyuria, prostatovesiculitis or a recent history of neisserian infection, acute epididymitis would seem more likely. With acute vasitis and funiculitis, palpation of the inguinal cord will reveal more tenderness than is found in most cases of torsion. Prehn's sign should be negative in epididymitis. McKay reported an acute torsion in the presence of a positive smear. A patient with acute epididymitis usually runs some fever with a moderate elevation of the white blood count.

Strangulated hernia. The symptomatology of acute strangulated hernia may be very similar to acute torsion of an inguinal testicle. The pain may be identical but the gastrointestinal symptoms will be less severe in torsion. Painful inguinal masses and swellings occurring in patients with undescended testes should make us suspect acute torsion. While the differential diagnosis may be most difficult in some cases, early surgical exploration is indicated in either case.

Torsion of the appendix testis. Torsion of the appendix testis occurs when this embryonal rest is pedunculated and becomes strangulated. Sudden severe pain may occur, but usually there is less swelling, edema, or testicular fixation. Palpation will reveal localized exquisite tenderness over the strangulated appendix testis.

Acute orchitis. Acute orchitis may follow or precede mumps or complicate a surgical

Fig. 1. Case 3. J.J., aged 23 years, admitted 4 months after recurrent swelling of the right testis showing small retracted gonad typical of neglected chronic torsion of the spermatic cord.

operation. These patients usually have fever, malaise, and acute pain in the involved testis. The testis will become enlarged, tense, and tender. There is never the fixation and enlargement of the spermatic cord in region of the external inguinal ring.

Neoplasm. Neoplasm of the testis is an insidious, slowly growing, relatively asymptomatic testicular enlargement, which usually is not confused with an acute torsion. There have been at least 8 reported cases in which a malignant tumor was found on microscopic sections following orchiectomy for torsion (Birdsall, Mohardt.)

Miscellaneous. There are other lesions as acute thrombophlebitis of the cord, filariasis, cellulitis, or trauma which may have to be considered in a few cases. In the presence of intra-abdominal undescended testes, lesions as acute appendicitis, acute diverticulitis, ureteral calculus, or acute seminal vesiculitis can cause diagnostic confusion. A careful history, physical and rectal examinations, urine analyses, and x-ray films will usually aid sufficiently to establish the correct diagnosis.

TREATMENT

Acute torsion of the spermatic cord should receive immediate surgical exploration. The length of time that a testicle will remain viable after torsion depends upon the amount of vascular constriction. In a patient in whom the artery as well as the vein has been suddenly occluded, the viability will be shorter than if only the vein is primarily occluded. While manual detorsion is always possible without surgery, the technique is unsafe and should be discarded. Surgical detorsion should always be followed by orchidopexy to preclude subsequent attacks. Ormond's case of a physician who practices manual detorsion for 12 years without testicular atrophy is an exception rather than the rule. Apparently this was a recurrent partial venous occlusion which could be satisfactorily reduced. Cahen operated upon a patient 12 hours after occlusion, found testicular gangrene necessitating orchiectomy. The practice of applying hot fomentation after detorsion is a good index of testicular viability. Surgical intervention within 8 hours will save the majority of acute torsions, but Birdsall states "in the majority of cases in which the diagnosis has been made early and the testicle found to be viable and detorsion performed, the testicle has undergone atrophy." Occasionally the strangulated tissue becomes infected resulting in gangrene.

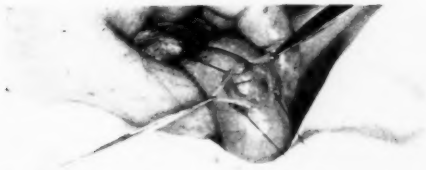

Fig. 2. Case 2. The remaining testis in recurrent bilateral torsion is sutured with interrupted silk to the dependent scrotal fascia.

In chronic recurrent cases the simple fixation of the lower pole of the testis to the dependent tunica vaginalis (Fig. 2) and fasciae will obviate subsequent acute attacks and gangrene. In all bilateral cases, the mate should be treated by fixation. Campbell has used the Jaboulay operation for the fixation of the opposite testis. In this technique the tunica vaginalis is everted to prevent a hydrocele, and the testis is anchored to the scrotum.

CASE REPORTS

Case 1. R.B., aged 20 years, while home on an overseas furlough on April 9, 1943, was suddenly seized during sleep with severe pain in the left testis.

Fig. 3. Case 1. R.B., aged 20 years, shows 1¼ clockwise turns to left producing aseptic gangrene of the testis. This patient went through several Southwest Pacific campaigns but not until he arrived home on an overseas furlough did he experience an acute torsion of the spermatic cord.

Within 2 hours the testis began to swell and a local physician was called. He admitted several recent sex exposures but denied any evidence of urethral discharge or history of venereal disease. A diagnosis of gonorrheal epididymitis was made and he was treated unsuccessfully with oral sulfonamides. On admission to Bushnell on May 9, 1945, examination revealed a slightly enlarged, firm, left testis and epididymis. The testis compared to the right seemed somewhat fixed within the upper scrotum. The remainder of the physical examination, x-rays, and laboratory tests were negative and noncontributory. A diagnosis of suppurative epididymitis was made and exploration on May 10, 1945, revealed marked peritesticular adhesions making delivery difficult. The mobilized testis was firm and irregular. The tunica vaginalis was opened and an intravaginal twist of 1½ clockwise turns was noted at the upper pole (Fig. 3). The tunica vaginalis was pale, fibrous, and slightly adherent to the tunica albuginea. Incision of the testis revealed testicular necrosis and a hemorrhagic distended epididymis. There was no bleeding. Removal of the cord and testis was carried out. The pathologic report showed aseptic necrosis and hemorrhage involving the entire testis and epididymis (Fig. 4). Following recovery patient was returned to duty.

It is interesting that this patient was fortunate to go through many front line battles unscathed and while at home in bed on an overseas furlough he suffered an acute undiagnosed lesion resulting in loss of one testis.

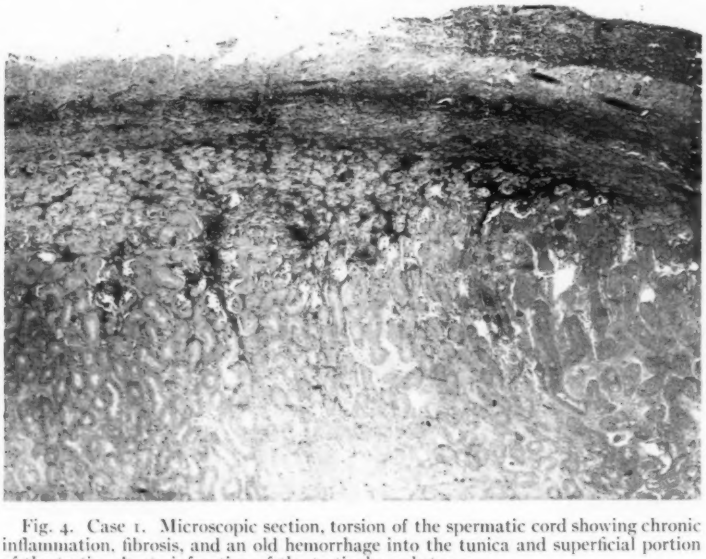

Fig. 4. Case 1. Microscopic section, torsion of the spermatic cord showing chronic inflammation, fibrosis, and an old hemorrhage into the tunica and superficial portion of the testis. Acute infarction of the testicular substance.

CASE 2. C.M., aged 22 years, gave a history of testicular injury at the age of 8 years. For the past 14 years he has had recurrent attacks of pain in each testicle, each attack lasting for 1 to 4 days. At 9:30 a.m. on July 1, 1943, he developed acute pain in the left testis. Later he developed pain in the left abdomen and swelling of the testicle. He was admitted to the station hospital and given morphine for persistent pain. After 4 weeks of symptomatic treatment he was transferred and admitted to Bushnell on July 28, 1943, with a diagnosis of orchitis. Examination revealed the right testis and epididymis to be normal. The left was enlarged 1½ times, hard, smooth, but not tender. There was slight enlargement of the left vas deferens and a small palpable nodule over the left pubic bone. The prostate and seminal vesicles were normal. The abdomen revealed no abnormalities, but was sentitive to deep palpation in the left lower quadrant. X-ray studies of the urinary tract, chest, and scrotum revealed no abnormalities. The blood count, urinalysis, and serology were within normal limits. The prolan A test of the morning specimen of urine was negative. Diagnoses of chronic torsion of the left spermatic cord with necrosis and chronic right recurrent torsion of the right spermatic cord were made. On August 11, 1943, under spinal anesthesia, the left testicle was delivered through a low left inguinal incision. Marked fibrous adhesions were noted surrounding the testicular coverings. The tunica vaginalis was yellowish-green and adherent to the tunica albuginea (Fig. 5). Upon dissection it was noted that the superior pole of the testicle presented a small, twisted, avascular pedicle with 1½ clockwise turns. The testicle was incised and there was no bleeding. The parenchyma was soft, putty-like, with a brown color. The left inguinal cord and testis were removed. The most dependent portion of the right scrotal sac was incised so as to include the tunica vaginalis. The exposed albuginea was sutured to the vaginalis and scrotal fascia with interrupted black silk (Fig. 2). Both incisions were closed with silk without drainage. The patient made an uneventful recovery and was returned to full military duty.

Fig. 5. Case 2. C.M., aged 22 years, aseptic necrosis of the left testis 6 weeks after acute torsion showing avascular dark brown putty-like testicular parenchyma.

Case of bilateral chronic recurrent torsion for 14 years which finally resulted in the loss of one testis. Bilateral orchidopexy should have been carried out many years before.

CASE 3. J.J., aged 23 years, 2 hours following sexual intercourse with an Alaskan Eskimo developed severe pain and swelling of the right testicle. In the hospital he was treated for acute epididymitis with oral sulfonamides. The swelling and soreness subsided after 18 days, but some induration remained. He remained well for 1½ years and in December, 1943, the tenderness and swelling recurred. He was again treated in the hospital for acute epididymitis. After 26 days, the swelling again subsided, but the right testis remained firmer than the left. He was returned to the continental United States with a diagnosis of neoplasm. On April 10, 1944, he was transferred to Bushnell. Examination of the urinary tract was entirely negative and the findings were limited to the scrotum. The right testis was smaller than its fellow and appeared rubbery hard and retracted into the external ring (Fig. 1). The left testis and epididymis were normal. The prostate and its secretion were normal, the urine was grossly clear. Laboratory examination of the urine, blood, and serology revealed no abnormalities. The prolan A test of the morning urine was negative. Preoperative diagnosis of chronic torsion of the right spermatic cord was made. On April 14, 1944 under local anesthesia, the right inguinal cord and upper scrotum were exposed. The testicular coverings were markedly adherent making delivery of the testis difficult. The testicle was small and rubbery in consistency with a firm area on the superior anterior pole representing the epididymis. The tunica vaginalis was very adherent and thin. Incision of the tunica albuginea revealed a greenish appearing testicle with a "cherry stem-like" pedicle. The pedicle above the upper pole had one complete counterclockwise turn, occluding the testicular blood supply. The cord was doubly clamped, ligated, and removed with the testis. Pathological examination showed a torsion of the spermatic cord with chronic inflamma-

Fig. 6. Case 3. Four months after acute torsion of the right spermatic cord the testis was small and on section showed an avascular greenish appearing parenchyma with a "cherry stem-like" pedicle.

tion and infarction of the right testicle and epididymis. Patient was returned to duty.

The sudden onset of acute testicular pain 2 hours following intercourse, in the absence of infection, should have suggested an acute torsion and immediate surgery.

Case 4. M.J., aged 22 years, in October, 1944, received a gunshot wound injuring the spinal cord at the level of sixth dorsal vertebra, resulting in immediate paralysis below that level. He received symptomatic treatment, and tidal drainage was begun with an indwelling urethral catheter. He was admitted to Bushnell on January 2, 1945, with a diagnosis of transverse myelitis, resulting in complete bowel, bladder, and leg paralysis. This patient was seen by the urologic service the first time on the day of admission and a well functioning urethral catheter was noted. The urine was cloudy and the original cultures showed diphtheroids, proteus, and Gram positive cocci unidentified. The cystometrogram made on February 2, 1945, showed a capacity of 450 cubic centimeters and a maximum voluntary pressure of 86 centimeters of water. On March 30, 1945, many bladder concretions were removed through a panendoscopic sheath. On May 26, 1945, he was seen again on account of swelling of the left scrotal sac. The left scrotal sac was red, swollen, but only slightly tender. The left vas deferens seemed indurated above the swelling. The indwelling catheter and tidal drainage were working well. Acute nonvenereal epididymitis was diagnosed and sulfadiazine, 1 gram four times daily, and penicillin 25,000 units, every 3 hours were initiated. This treatment was continued for 15 days without noticeable improvement. On June 13, 1945, an exploration of the left scrotal contents was carried out under intravenous pentothal anesthesia. The testis was delivered with difficulty due to fibrous adhesions involving all the structures around the left testicle. With sharp dissection, the testis was delivered and opening of the tunica vaginalis showed aseptic necrosis. The vascular pedicle was anomalous, twisted ¾ clockwise turn and attached to the upper pole of the testis. The entire mass including the left spermatic cord was removed. The microscopic sections showed old and recent thrombi involving arteries and veins of the spermatic cord with aseptic necrosis of the left testis and epididymis. The wound healed *per primam*. The right testicle has remained normal.

This patient has a complete paraplegia, but contractions of the cremasteric muscle are preserved and occur while the patient has "abdominal spasms." This is the first recorded case of torsion occurring in a total paraplegia and suggests segmental cremasteric contractions. Acute torsion was not considered in the differential diagnosis on the first visit.

Case 5. H.B., Negro, aged 20 years, had swelling and pain in the left scrotal sac in 1941 which lasted for 1 day. He had a similar attack in 1942 while walking to work. An attack of acute gonorrhea in 1943 was cleared in 2 weeks without complications. While lying down on an island overseas on May 20, 1945, he experienced an acute excruciating pain in the left scrotal sac followed by swelling. He was admitted to the station hospital and treatment for acute epididymitis was begun. He failed to improve and was transferred to Hawaii and admitted to the 318th General Hospital on June, 8, 1945. Admission examination revealed the left testis three times the size of the right, smooth, firm, and what seemed to be an indurated epididymis. The skin overlying the left scrotal contents was definitely adherent anteriorly. Intravenous pyelograms and chest film were normal. The urine was clear and negative for tubercle bacilli. The tuberculin skin test showed 1+ positive. The administration of 200,000 units of penicillin failed to reduce the swelling. Chronic torsion was now suspected. On July 5, 1945, exploration of the left scrotal contents revealed marked peritesticular adhesions, intravaginal torsion ½ counterclockwise turn, "cherry stem" vascular pedicle and yellow-green tunicae. Incision of the testis showed no bleeding. The testicle and cord were removed. The microscopic sections showed complete aseptic necrosis of the testicle and epididymis due to spermatic cord torsion. Following the operation recovery was uneventful.

Acute torsion occurring in a soldier under fire resulting in an improper immediate diagnosis, neglect, and loss of one testicle. The correct diagnosis should have been suspected from the history (C.J.S.).

SUMMARY

An anomaly of the testicular attachments and the vascular pedicle is necessary for torsion of the cord to take place. The precipitating factors are the intravaginal and extravaginal fibers of the cremasteric muscle. Muschat has shown that, when the tunica vaginalis abnormally invests and encircles the entire epididymis, the testis and epidydymis will not be attached to the scrotal wall. In these cases the testicular blood supply enters the upper pole. A polar pedicle with a high investment and a relaxed tunica is an ideal anomaly for an acute torsion. Why and how the contractions of the cremasteric muscles are initiated is still not well understood. It is understandable how muscular effort, direct or indirect, may influence its contractions but it is less understandable when torsion occurs during periods of physical relaxation or sleep.

It is of unusual interest to note the case of acute torsion in complete transverse myelitis (Case 4). Segmental cremasteric muscle contractions of the lateral and median fibers may be observed in this patient's scrotum. In the presence of an anomalous testicular pedicle unco-ordinated segmental spasms and contractions of the cremasteric muscles could readily precipitate and maintain an acute spermatic cord torsion until tissue edema and swelling supervene. Goodrich suggested the counterspin of the testis was an important factor in acute torsion. This seems, in part, to be the *modus operandi*, as only a few cases undergo spontaneous detorsion. In cases of mild recurrent torsion there is only sufficient testicular mobility to cause passive congestion and pain. These mild cases may reduce themselves or are reduced by the patient.

The record in the management of spermatic cord torsion leaves much to be desired. The early signs and symptoms are still unrecognized and ignored. Masterly inaction instead of immediate surgery still results in the loss of 75 per cent of the testicles in patients operated upon late. If in the differential diagnosis of every inguinal and scrotal swelling characterized by severe sudden pain, torsion were considered, many more cases would be diagnosed early. The history itself many times suggests the correct diagnosis. Acute testicular pain following any conceivable type of muscular exertion, work or play, should forever raise the question of torsion in the examiner's mind. Torsion occurring during sleep should be more widely recognized and followed by prompt surgical exploration. In all fairness, the fault is not always on the side of medical attendants. Parents and patients refuse early surgery in approximately 15 per cent of cases.

Many testicles could be saved if we would explore a few cases of acute epididymitis for a mistaken torsion rather than treat the majority of acute torsions conservatively as epididymitis. Acute epididymitis treated surgically usually recovers while torsion treated by inaction and conservatism will result in fibrosis and complete testicular atrophy.

CONCLUSIONS

1. Acute torsion of the spermatic cord is frequently undiagnosed, and after its occurrence only 1 in 5 testes are saved.

2. Intravaginal torsion in descended testicle occurs when an anomaly of the testicular attachments and pedicle is present, precipitated by segmental contraction of the cremasteric muscle.

3. Chronic torsions of the spermatic cord should be anchored surgically to the dependent scrotum before the acute emergency occurs.

4. Acute torsion operated upon immediately or within 8 hours will usually result in a viable organ.

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EXPERIMENTAL STUDY ON LIVER FUNCTION TESTS

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THERE is at present a tendency to perform simultaneously several tests of liver function and to repeat them during the course of an illness since this is the only way of obtaining fairly accurate data concerning the functional state of the organ. This tendency, perfectly justified by the studies of experimental hepatic pathology and recently confirmed by studies with patients (24, 25, 26, 28, 30, 35), is based on the following facts:

1. The liver is characterized by its multiple functional activities and these may be variously affected by a certain type of lesion. Thus "the employment of a number of tests in different phases of the disease will lead to a satisfactory appraisal of the physiologic status of the organ" (23).

2. "The hepatic function varies not only from day to day but from hour to hour" (Ivy). In this statement are implied the concepts of duration of illness and of hepatic regeneration. The time element (duration of illness) is important as regards hepatic regeneration. The liver's great capacity for regeneration is a factor that limits the use of liver function tests. "Unless the hepatic injury be extensive and rapid the simultaneous incidence of regenerative processes can compensate and overshadow the destructive effect of the pathological processes" (21). We were able to confirm this assertion (4, 5).

3. A close interrelation exists among the intrahepatic and extrahepatic (biliary tract) pathological processes. Snell and Magath pointed out accurately that clinically a gross extrahepatic biliary obstruction does not exist without injury of the hepatic parenchyma, nor a pure form of jaundice without injury or obstruction of the finer bile ducts or canaliculi. This fact must be taken into account when the purpose of liver function tests is that of obtaining data for the differential diagnosis of jaundice.

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As Ivy (21) has pointed out, the purposes of performing liver function tests can be summarized in three main objectives: (a) to establish whether or not a hepatic disturbance exists, (b) to establish a prognosis of the disturbance and to aid in determining the surgical risk, (c) to determine the differential diagnosis of jaundice.

Drill and Ivy (14) made a comparative experimental study of five liver function tests. They used dogs to which they administered twice a week small doses of carbontetrachloride, a hepatotoxic drug. The purpose of their study was to investigate the comparative sensitivity of the tests in three respects: (a) capacity to discover the hepatic damage, (b) continued validity of the tests as the administration of carbontetrachloride was prolonged, and as well (c) the existence of association or dissociation among the several liver function tests.

With an almost similar purpose we, coincidentally and independently, have made a comparative experimental study of the prothrombin concentration of plasma, the bilirubinemia, and the acid and alkaline phosphatase activity of the serum of dogs in which different degrees of hepatic damage were produced: (a) by chloroform anesthesia, (b) by cholecystectomy and ligation of the common duct, and (c) by cholecystectomy. Cholecystectomy alone was chosen as a procedure that produces, in dogs, modifications of the phosphatasemia not attributable merely to a postoperative reaction but apparently due to a determined though slight degree of biliary insufficiency (12).

METHODS

1. *Chloroform anesthesia.* Normal dogs which had been fasted for 3 days (water at will) were used. They were subjected to a

TABLE I.—RESULTS, CHLOROFORM ANESTHESIA

Dog	Days	Phosphatase		Bilirubin mgm. per 100 ml.	Prothrombin %
		Alkaline unit per 100 ml.	Acid unit per 100 ml.		
343	Initial	3.05	0.42	0.58	103
	1	4.94	1.42	0.76	23.6
	2	16.5	1.06	0.63	17.5
	3	16.2	0.41	0.68	30
	6	14.6	0.50	0.79	100
	8	13.5	0.72	0.66	—
	10	8.1	0.61	0.68	—
	13	2.5	0.41	0.60	—
353	Initial	2.5	0.9	0.55	92
	1	9.5	1.01	1.26	36.7
	2	17.8	1.01	1.98	6.5
	3	18.6	1.01	0.71	18
	6	13.5	0.87	0.82	92
	8	11.9	0.45	0.70	—
	10	9.4	0.81	0.76	—
	13	7.8	0	0.80	—
	15	6.5	0.41	0.71	—
	17	5.7	0.41	0.70	—

chloroform anesthesia of 1 hour's duration; a Boyle apparatus was used. According to long experience acquired in this laboratory the procedure produces a hepatic lesion (hyaline central necrosis) which is followed after 3 weeks by a complete histological recovery of the parenchyma, although regeneration by rapid multiplication of the remaining hepatic cells replaces the greater part of the functional deficit of the organ within 6 days (3, 36). The determinations of prothrombinemia, bilirubinemia, and acid and alkaline phosphatasemia were performed at variable intervals from 3 hours to as long as 17 days following the anesthesia.

2. *Cholecystectomy and ligation of the common duct.* Normal dogs were used and operated upon under ether anesthesia. The operation was performed aseptically, the gall bladder was removed, and the common duct severed and tied. Care was taken to perform an impeccable surgical operation in order to reduce to a minimum the reactions caused by the operation itself. The first determinations were made 15 hours after the operation and repeated at variable intervals during the survival period of the animals (from 3 to 212 days). In every instance an autopsy was performed in order to check any possible recanalization of biliary passages. All the animals were kept on a diet consisting of bread, milk, and syrup (6).

3. *Cholecystectomy.* Cholecystectomy was performed on normal dogs, under ether anesthesia. The first determinations (bilirubinemia and acid and alkaline phosphatasemia) were performed 72 hours after the operation and periodically repeated until the 50th day.

The phosphatasemia was determined by Bodansky's method modified by Shinowara, Jones and Reinhart (32) which simultaneously measures the acid and alkaline phosphatase activity. The unhemolyzed serums were tested within 24 hours of obtaining the samples. In the preparation of the substratum glycerophosphate B. D. H. and sodium veronal were employed. The *p*H was determined by means of a Beckman potentiometer. The final valuation of the phosphate anion was made photometrically with a Leifo photometer. The results are expressed in units per 100 milligrams of serum. The unit equals 1 milligram of phosphorus (as phosphate ion) liberated during 1 hour of incubation at 37 degrees C., sodium glycerophosphate being used as a substratum (without the hydrolysis exceeding 10 per cent of the substratum) and at optimum *p*H of 5.00 ± 0.15 for the alkaline phosphatase. By this method the normal values in dogs oscillate between 1.8 and 7.4 units per 100 milligrams (alkaline) and from 0 to 1.1 units per 100 milligrams (acid).

The total bilirubin of the serum was determined by adapting to the photometer the method of Ernst and Forster (16). Redistilled acetone was used as the reagent for the extraction. After this was added to the serum the covered tubes were kept for 15 minutes in the icebox and then centrifuged. Normal values for dogs by this method are 0 to 0.7 milligram of bilirubin per 100 milligrams of serum.

The prothrombin concentration of plasma was estimated according to the method of Tanturi and Banfi (34). The values were expressed in a per cent of the normal values,

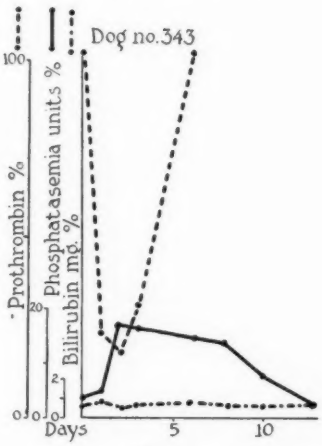

Chart 1.

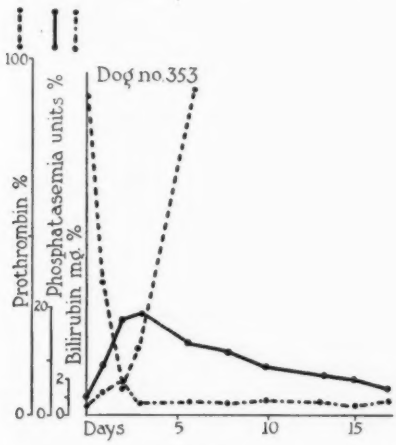

Chart 2.

according to the average of results obtained from normal dogs.

RESULTS

1. Chloroform anesthesia. From Table I and Charts 1 and 2 it can be seen that:

a. Increase in alkaline phosphatase and decrease in prothrombinemia are more sensitive indices than is bilirubinemia in detecting the hepatic damage produced by chloroform anesthesia.

b. Hypoprothrombinemia is an earlier index of hepatic damage than is hyperphosphatasemia. It reaches its maximum during the 24 to 48 hours following the anesthesia while the highest value of the phosphatasemia is recorded between 48 and 120 hours following the anesthesia.

c. At the 5th day (or at most, the 6th) the prothrombinemia has reverted to its normal value while the phosphatasemia remains above the normal level until the 10th to the 13th day.

d. The value of the bilirubinemia is not greatly modified, ascending slightly and reaching its maximum between 24 and 48 hours, and then reverting to normal. Compared with prothrombinemia, bilirubinemia gives no information regarding the severity of damage.

2. Cholecystectomy and ligation of the common duct. Tables II and III, and Charts 3, 4, and 5 show that:

a. The increase in serum alkaline phosphatase is greater than that in the experiments of chloroform intoxication (one hundred and fifty times the normal initial level). Fifteen hours after operation the increase is very pronounced, but the maximum value is reached at some point between the 4th and 94th day. Then an irregular and constant decline begins which lasts until the death of the animal. This decrease may reach a value smaller than the initial increase, but is always above the normal level.

b. The acid phosphatase activity parallels the alkaline but to a lesser extent. This increase is slightly greater than in the case of chloroform intoxication.

c. Bilirubin increases after the ligation of the common duct. The maximum concentration is obtained between the 4th and the 52d day following operation. Afterward it stabilizes at a level slightly above the normal. In one experiment (Dog 375, Chart 5) a sudden decrease to a 0 level can be observed. The autopsy findings revealed a spontaneous rupture of the biliary passages.

d. Prothrombinemia is late in revealing hepatic damage due to obstruction; only at the final period of the experiment is its value consistently low.

3. Cholecystectomy. Table IV shows:

a. The alkaline phosphatase increases in the cholecystectomized dogs; the increase is great-

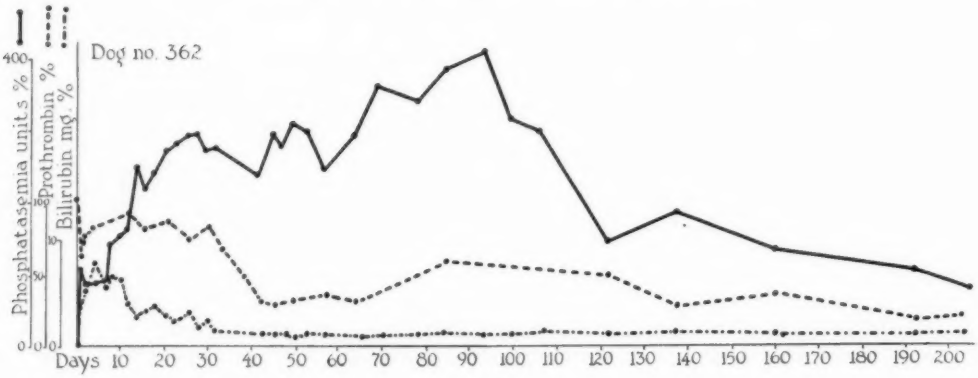

Chart 3.

er than in the chloroform experiments but less than in experimental biliary obstruction. The maximum level is observed between the 10th and the 16th day following operation. After this period a decline begins and the normal level is reached at the end of the 50th day.

b. The bilirubinemia does not suffer any variation.

c. The prothrombinemia was not studied in these experiments since previous work done in this laboratory demonstrated that in cholecystectomized dogs it changes only slightly during the first 24 hours following operation. This change is not due to the cholecystectomy itself since the same variations were observed in dogs in which all the surgical steps, except the removal of the gall bladder, were repeated (3–6). The slight variations in the prothrombinemia do not last more than 24 hours and are attributed to the anesthesia and section of the tissues.

COMMENT

The alkaline phosphatase is present normally in the blood serum and its increase has been related to different types of liver injury (1, 2, 8, 9, 15, 18, 19, 29); therefore it was considered a sensitive indication of the hepatic damage (in absence of bone lesions). Several studies (9, 27) seem to indicate that the liver

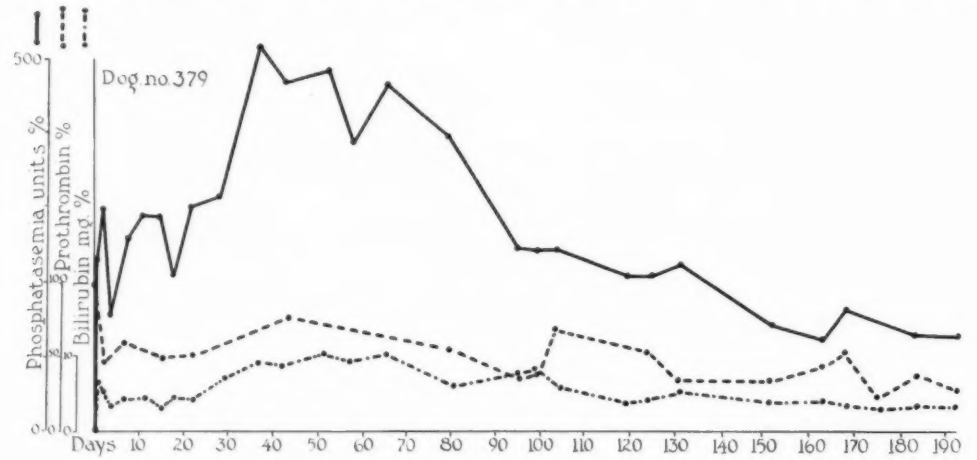

Chart 4.

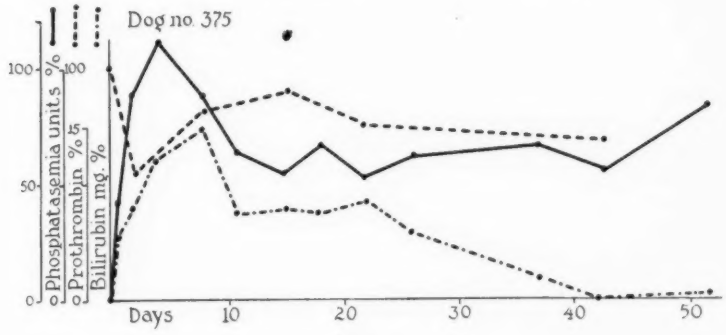

Chart 5.

is an important source of this enzyme. It is almost certain that the alkaline phosphatase excreted in the bile is of hepatic origin (15, 17).

Drill and associates (13) claim that the increase of the alkaline phosphatase activity of serum accompanying the hepatic lesions is related to an alkaline phosphatase normally present in the blood in very small quantities and this might be the alkaline phosphatase II, which was extracted from the liver tissue by Cloetens.

Kabath obtained the release of the enzyme through cellular autolysis. Sharnoff, Lisa, and Riedel assert that the injured cells release phosphatase into the blood; consequently the degree of the injury regulates the enzyme level reached in the blood. Any agent able to produce necrosis or necrobiosis of the hepatic cells of the middle of the lobule (which contain phosphatase and apparently are the earlier

TABLE II.—RESULTS, CHOLECYSTECTOMY AND LIGATION OF COMMON DUCT

Dog	Days	Phosphatase		Bilirubin mgm. per 100 ml.	Prothrombin %
		Alkaline unit per 100 ml.	Acid unit per 100 ml.		
302	Initial	3.26	0.52	0.55	101
	1	105.6	1.80	0.58	63
	2	81.9	1.65	8.77	77
	4	90.9	0.41	11.52	83
	8	137.9	1.02	9.60	—
	12	161.9	1.21	6.25	93
	16	217.6	1.44	5.10	82
	21	272.8	1.83	4.33	86
	26	289.1	1.64	4.65	74.5
	30	272.9	1.01	3.28	80
	45	289.5	1.22	1.70	30.6
	50	305.7	1.43	1.50	32.8
	57	240.7	1.43	1.70	35.6
	64	295.7	2.24	1.20	30.9
	85	371.2	1.43	1.92	57.1
	94	404.3	1.42	1.75	—
	107	290.2	1.64	1.92	—
	122	143.5	1.90	1.55	48
	137	183	1.60	1.98	27
	160	127.7	0.91	1.70	35
	192	101.3	1.23	1.71	18
	204	78.2	1.02	2	20

TABLE III.—RESULTS

Dog	Days	Phosphatase		Bilirubin mg. per 100 ml.	Prothrombin %
		Alkaline unit per 100 ml.	Acid unit per 100 ml.		
375	Initial	1.43	0.61	0	101
	15 hours	68.3	1.84	5.12	—
	2 days	176.5	2.66	8.01	52.7
	4 days	222.8	0.83	12.2	—
	8 days	174.7	1.03	14.82	82.6
	11 days	125.9	0.81	7.68	—
	15 days	109.7	0.82	8.01	90.1
	18 days	132.5	1.84	7.68	—
	22 days	108.5	1.23	8.54	75.8
	28 days	120.4	1.42	6.08	—
	37 days	136.2	1.02	2.24	—
	43 days	113.8	1.03	0	69.4
	52 days	169.1	1.42	0.58	—

TABLE IV.—RESULTS, CHOLECYSTECTOMY

Dog	Days	Phosphatase		Bilirubin mg. per 100 ml.
		Alkaline unit per 100 ml.	Acid unit per 100 ml.	
581	Initial	3.2	0.8	0
	3	21.2	0.8	0
	10	45.4	0	0
	16	50.5	0	0
	24	33.8	0	0
	34	29.3	0.88	0
	50	6.3	0	0
585	Initial	2.7	0.9	0
	3	7.0	—	0
	10	19.8	0.8	0
	16	12.9	0.4	0
	24	9.9	0.45	0
	34	8.85	0.72	0
	50	2.52	0	0

sufferers of the injuries) will produce an abnormal release of the enzyme into the blood. Transfusion of dog's icteric blood (which contains increased alkaline phosphatase) to a normal dog, increases the blood phosphatase of the recipient, and this increase persists a considerable time after the bile pigments are excreted (15).

Our experiments show that the highest levels of blood alkaline phosphatase are obtained following experimental extrahepatic biliary obstruction. In hepatitis due to chloroform an increase is found which persists even after the prothrombinemia has reached its normal level. This increased phosphatasemia, which lasts only a few days, can be explained by the slow excretion of the excess phosphatase released during the period of the maximum injury produced by chloroform (48 hours). The protein structure of phosphatase makes it unexcretable by the kidneys (17), and the persistence of an alkaline phosphatasemia above the normal when the prothrombinemia has reached its normal level (chloroform hepatitis) might well be interpreted as an indication of a biliary obstruction or hepatic lesion which is recovering. In other words, even accepting that normal excretion of hepatic phosphatase is in the bile, once the enzyme reaches the blood either by cellular lysis or by the bile pouring out into circulation the phosphatase can not be re-excreted through the biliary passages even if these have been completely restored. The experiment of the transfusion of icteric blood with increased phosphatase to a normal dog (15) referred to above warrants that conclusion. If this interpretation is correct, the determination of the alkaline phosphatasemia as a test of liver function would be submitted to a limitation which should be considered, especially if it is used as a *single* liver function test. In this latter case, the hyperphosphatasemia might denote a hepatic damage which had existed before. The doubt could only be cleared up by repeating the determination on succeeding days and by comparing the results with the results of other liver function tests. Even so, the comparative values will depend on the reliability of the tests chosen for the comparison.

In the work of Drill and Ivy (14) in studying the comparative sensitivity of tests, it was shown that the bromsulfalein test was the most sensitive in detecting hepatic damage. All the animals, except one, had hyperphosphatasemia at the same time or slightly after the bromsulfalein clearance had reached the normal values. Obviously the phosphatasemia was as sensitive as the bromsulfalein test. The prothrombin time was not so sensitive as the above mentioned tests, though it detected the hepatic damage prior to the galactose tolerance test.

In our experiments the prothrombinemia was the most sensitive test in detecting the hepatic injury produced by chloroform because it preceded the increase of the blood alkaline phosphatase. The results of Drill and Ivy are disputable concerning the prothrombin technique used by them. The difference in our results when compared with those of Drill and Ivy, cannot be accounted for by the different hepatoxic drug used by us (chloroform) because it was demonstrated (3) that chloroform produces the same variations in prothrombinemia as does the carbontetrachloride in similar experimental conditions. Our results show that prothrombinemia detects earlier than the phosphatasemia the hepatic damage produced by chloroform, and

its variations closely indicate the course of the injury as well as the reactive capacity of the liver. As has been said, phosphatasemia persisted at a high level even after prothrombinemia had reached its normal value and the high level was not the expression of a lesion in activity but in regression.

We have mentioned that the highest values of phosphatasemia are obtained in experimental extrahepatic biliary obstruction. It seems that phosphatasemia is a very sensitive index of biliary obstruction, either intrahepatic or extrahepatic, if the experimental results of ligation of the common duct and those of cholecystectomy are compared.

In extrahepatic biliary obstruction there are two factors to be considered: (a) the pouring off of bile (which contains the phosphatase secreted by the uninjured cells of the liver as well as other elements) into the blood stream; (b) the release of phosphatase directly into the blood by the injured cells. This explains the highest values of bilirubinemia and phosphatasemia found in this instance. The bile pigments are excreted through the kidneys, and this excretion accounts for the steady blood level that follows the initial increase (dogs 362 and 379) except when a rupture of the biliary tract (dog 375) suddenly dropped it to a 0 level. On the contrary the kidney is impermeable to phosphatase (17) and this persists at a high level in the blood nearly until the end of the experiment when the hepatic damage is severe and the hepatic insufficiency is at its height as is revealed by the marked hypoprothrombinemia. It is probable that at this moment the liver cells are deprived of phosphatase though its lysis does not increase the enzyme level in the blood (31).

In experimental biliary obstruction prothrombinemia is late in denoting the hepatic damage; only in the final period of the experiment is its value really low. These results confirm our earlier experiments (4) on experimental biliary obstruction in which it had been shown that prothrombinemia does not suffer significant variations (hemorrhagic levels) during the survival period of the animal, and that the hypoprothrombinemia and hemorrhagic tendency observed in the clinical cases of obstructive jaundice are due to the injury of the hepatic cells, which complicates or follows the biliary obstruction, and is not due to the mechanical disturbance of the intrahepatic biliary stasis.

Special mention of the behavior of the acid phosphatase is necessary. In experimental biliary obstruction acid phosphatase does not vary in the same magnitude as does the alkaline; however, it increases and this increase is slightly higher than that observed in the chloroform experiments. In experimental biliary obstruction acid phosphatasemia parallels alkaline phosphatasemia variations although in a much smaller proportion. No explanation of this fact can be offered based on our present knowledge.

The results of the experiments of cholecystectomy revives the problem of cholecystectomy and dilatation of biliary ducts and hepatitis (7).

Cantarow (10) reported that cholecystectomy in man temporarily decreases the bromsulfalein excretion when the test is performed 24 hours following operation. Bergh (7) did not find functional hepatic changes in cholecystectomized dogs from 1 week to 15 months following operation; they used the bromsulfalein at a dose of 2 milligrams per kilogram of body weight, the van den Bergh reaction and the icterus index as liver function tests. As it was recently shown that the bromsulfalein test at a dose of 5 milligram per kilogram body weight is a more sensitive index, and that phosphatasemia is also a very sensitive index of the hepatic injury (14) Drill, Annegers and Ivy (12) restudied the effects of cholecystectomy on the hepatic function in dogs by means of both tests. Ten of 12 cholecystectomized dogs showed an increased phosphatasemia following the operation and in 9 of them this increase lasted as long as 70 days—the entire period of observation. Four of these 10 dogs showed an increased bromsulfalein retention. Drill, Annegers, and Ivy explain the hyperphosphatasemia observed following cholecystectomy in dogs on the basis of the experimental finding that cholecystectomy produces a dilatation of the bile ducts (7) and an incompetency of the sphincter of Oddi which permits transmission of the intraduodenal pressure into the biliary tract (20). They

were not surprised by the long lasting increased phosphatasemia after cholecystectomy though they considered it to be a sensitive index of the slight functional biliary obstruction connected with the dilatation of the intrahepatic bile ducts.

Our results confirm those of Drill (12) concerning the increase of the blood alkaline phosphatase following cholecystectomy in dogs. Although our studies were performed on only 2 dogs, the results closely coincide and warrant definite conclusions. However, in our animals the phosphatasemia returned to normal at an earlier period. This return to normal cannot be explained nor can the mechanism by which the functional changes of the intracanalicular pressure causes the phosphatasemia to rise.

The bilirubinemia does not change following cholecystectomy in dogs and this is not surprising if the great excretory capacity of the liver for bilirubin is recalled. Moreover, the increase of phosphatasemia is not necessarily associated with jaundice (15), and this is clearly shown in our cholecystectomy and chloroform experiments. These emphasize the concept of the existence of hepatic injuries which are not revealed by variations of the bilirubinemia.

Our comparative results show the existence of liver function tests which are very sensitive in their capacity to detect the hepatic damage, the course of the lesion, the reactive capacity of the liver, and the minimal biliary obstruction.

We have not attempted to evaluate the different tests as aids in determining the differential diagnosis of jaundice because, as is known, the dog is not an experimental subject for that purpose since he requires very extensive and severe hepatic damage or a total biliary obstruction to produce a degree of jaundice of certain intensity. However, this does not nullify the value of the results obtained from the comparative study of the bilirubinemia with the other tests.

In spite of the limitations imposed by the fact that it is not always possible to apply the results of animal experimentation to the human, our experiments demonstrate that: (1) prothrombinemia is more sensitive than phosphatasemia in detecting cellular hepatic lesions, the course of the lesion, and the reactive capacity of the liver against injury; (2) phosphatasemia is more sensitive than is prothrombinemia in detecting the minimal biliary obstruction, i.e. one which could be designated as *functional biliary obstruction*. These statements apparently suggest the idea of the existence of a dissociation of the liver function tests which obviously implies the concept of a dissociation of the functions of the liver (metabolic and excretory). This is not erroneous so far as it is concerned with the reserve power of the liver. However, in accepting this idea one is forced to consider the existence of a systematized pathologic histology of the liver (pure forms of cellular injury and pure forms of canalicular injury) which, as is known, does not agree with the clinical reality.

The evidence points out that a given degree of hepatic injury does not equally affect the different functional activities of the liver in a quantitative manner, and, in order to gain knowledge of the course of any kind of hepatic damage, it is necessary to perform a series of different liver function tests. Therefore, the above concept should be replaced by the concept of quantity of injured parenchyma or by that of lesser or greater sensitivity of the liver function tests.

Despite the concept brought out by the experimental hepatic pathology that a minimal quantity of healthy liver parenchyma can accomplish some functional activities as normally, the analysis of our results, *considering the liver as a whole*, demonstrates that prothrombinemia detects earliest the damage of the hepatic cells, but that phosphatasemia is more sensitive in revealing biliary obstruction. As the severity of the damage increases both tests become abnormal and probably other less sensitive tests do also. This concept is in accordance with the idea of Ivy (14) of a *quantitative association* of the liver function tests instead of a *qualitative dissociation* of the liver's functions.

CONCLUSIONS

1. Chloroform anesthesia of 1 hour's duration produces in the dog a hypoprothrombine-

mia and an increase of the alkaline phosphatase in the blood. The decrease in the prothrombin concentration precedes the hyperphosphatasemia and it reaches its maximum during the 24 to 48 hours following the anesthesia, while the highest value of the phosphatasemia is recorded between 48 and 120 hours following the anesthesia. By the 5th or the 6th day the prothrombinemia has reverted to its normal value while the phosphatasemia remains above the normal level until the 10th to 13th day.

The value of the bilirubinemia is not greatly modified, ascending slightly and reaching its maximum between 24 to 48 hours and then reverting to normal.

All these changes are closely related to the *degree* of the hepatic injury produced by chloroform.

2. Cholecystectomy and ligation of the common duct produce in the dog an increase of the serum alkaline phosphatase greater than in the experiments of chloroform intoxication (150 times the normal initial level). Fifteen hours following the operation the increase is very pronounced but the maximum value is reached at the end of a variable period which oscillates between the 4th and the 94th day. Then an irregular but constant decline begins and this decline lasts until the death of the animal.

The acid phosphatase activity parallels the alkaline but not in the same proportion. This increase is slightly greater than in the chloroform intoxication.

The prothrombinemia is late in revealing the hepatic damage due to the obstruction; only in the final period of the experiment is its value consistently low.

The bilirubinemia increases after the ligation of the common duct. The maximal concentration is obtained between the 4th and the 52nd day following the operation. Afterward it stabilizes at a level slightly above the normal.

3. Cholecystectomy alone produces an increase of the alkaline phosphatase; the increase is greater than in the chloroform experiments but less than in the experimental biliary obstruction. The maximum level is observed between the 10th and the 16th day following the operation. Afterward a decline begins which reaches the normal level at the end of the 50th day.

The bilirubinemia does not suffer any variation.

The prothrombinemia suffers very slight variations during the first 24 hours after the operation and it is not related to the cholecystectomy itself.

4. A comparative study of the results demonstrates that prothrombinemia is more sensitive than phosphatasemia in denoting the cellular hepatic lesion. The phosphatasemia is more sensitive than is prothombinemia in detecting minimal biliary obstruction; i.e. one which could be designated as a functional biliary obstruction. As the severity of the damage increases, both tests become abnormal. It is concluded that it is more reasonable to accept the concept of a quantitative association of the liver function tests rather than of qualitative dissociation of the liver's functions.

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SURGICAL TREATMENT OF PANCREATIC CYSTS

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PANCREATIC cyst is a disease of infrequent occurrence, although it is encountered often enough to merit some consideration in regard to accurate diagnosis and effective treatment. A review of the accumulated clinical records reveals that this diagnosis has been made in only 9 cases since the origin of the Lahey Clinic in 1926 to October 1, 1945.

Pancreatic cysts are usually observed in patients in middle life. They have been found to be present, however, at an early age, 1 case being reported in a 5 month old child (5). According to several reports in the literature, the two sexes are affected with equal frequency. (6, 18). In the present series of 9 cases there were 6 females and 3 males. The youngest was 18 and the eldest 71 years of age.

Robson and Cammidge classified pancreatic cysts into the following groups:

1. *Retention cysts*, which are lined with epithelium and are caused by obstruction in the pancreatic duct, smaller ducts, or acini.

2. *Proliferation cysts*, the result of proliferation of glandular epithelium followed by an accumulation of fluid. These are tumor formations (cystadenomas).

3. *Congenital cysts*.

4. *Hemorrhagic cysts*, secondary to necrosis and bleeding.

5. *Hydatid cysts*.

6. *Pseudocysts*, produced by trauma or degenerative changes of the interstitial tissue of the pancreas. These are distinguished from true cysts in that they are not within the substance of the pancreas but outside it and usually lie in the lesser omental sac. They have no epithelial lining.

7. *Dermoid cysts* (added by Judd, 12).

A summary of the types of pancreatic cysts which are found in this series is presented in Table I.

In the earlier literature, trauma was frequently mentioned as the etiologic agent in the production of pancreatic cysts (10, 16, 17, 23). No accurate estimation of the percentage of frequency with which trauma was a causative factor can be made. In this group of cases, history of trauma was elicited in only 1 case.

More recently, the rôle of acute and chronic pancreatitis as the etiologic agent has been emphasized (12, 18). The pathogenesis is still undecided. It has been suggested that partial obstruction of the pancreatic ducts may lead to necrosis of the tissues by the pancreatic enzymes (4, 24). Archibald believed that pancreatitis may be the result of bile entering the pancreatic duct. In 3 cases of the present group, there was associated disease of the biliary tract. There was a previous history of jaundice in 3 of the cases. One patient had undergone elsewhere an exploratory laparotomy for acute cholecystitis; in another case, cholecystostomy had been done previously. Gallstones were noted in 2 of these cases at operation (Table II).

ANATOMY

An anatomical study shows the pancreas lying in the epigastrium and left hypochondrium in a retroperitoneal position, behind the serosal floor of the lesser omental sac, at the level of the first and second lumbar vertebrae. It is bounded posteriorly by the nonresilient posterior abdominal wall, and for this reason lesions of the pancreas are likely to extend forward in the direction of least resistance, and usually into the lesser sac. A diagrammatic sagittal section through the body of the pancreas demonstrates more clearly the various locations in which a pancreatic cyst may be found (Fig. 1).

The three most common locations occupied by pancreatic cysts are listed in order of frequency, according to Judd, Mattson, and Mahorner (13).

1. Cyst presenting between the stomach and transverse colon, and behind the gastrocolic ligament.

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TABLE I.—TYPES OF PANCREATIC CYSTS

Case	Sex	Age, years	Type of cyst
1	F	43	Papillary cystadenocarcinoma
2	F	71	Pseudocyst
3	F	58	Hemorrhagic cyst
4	F	39	Pseudocyst
5	M	45	Pseudocyst
6	F	51	Papillary cystadenocarcinoma
7	F	41	Pseudocyst
8	M	18	Pseudocyst
9	M	32	Pseudocyst

2. Cyst protruding into the lesser sac and presenting between the stomach and liver.

3. Cyst lying between the layers of the mesocolon, either behind the transverse colon or below it.

Rarely, these cysts may assume a position behind the descending colon, and may even descend into the pelvis.

In this series, the pancreatic cyst presented between the stomach and liver in 2 cases; posterior to the stomach and pressing against the gastrohepatic omentum in 2 cases; along the lesser curvature of the duodenum in the head of the pancreas in 3 cases; along the lesser curvature of the stomach in 1 case, and at the root of the mesocolon in 1 case.

DIAGNOSIS

Pain is the most frequent symptom. It is usually experienced in the right hypochondrium and epigastrium, and may be referred to the back. Its intensity varies from a dull ache to a fairly severe pain requiring sedation. Frequently there may be only symptoms of pressure on the neighboring organs, especially on the stomach, with anorexia and epigastric discomfort after meals, which may be accompanied by nausea and vomiting and gaseous distention.

The presence of an abdominal mass situated above the level of the umbilicus, usually in the left hypochondrium, is the most helpful and reliable diagnostic sign. The surface of the cyst is likely to be smooth, tense, and immobile, except for those cysts located in the tail of the pancreas. Unless such a mass is palpable or demonstrable by roentgenographic study, a diagnosis of pancreatic cyst is difficult or impossible.

Roentgenologic examination is a major aid in the diagnosis of cysts of the pancreas. The presence of an enlarged duodenal loop, or a filling defect of the stomach, duodenum, or transverse colon produced by an extrinsic mass is evidence in support of a presumptive diagnosis of a pancreatic cyst.

Since a pancreatic cyst may extend into any part of the abdominal cavity, it must be distinguished from a distended gall bladder, omental or mesenteric cyst, hydronephrosis, a retroperitoneal tumor, an ovarian cyst, and cysts or tumors of the liver.

SURGICAL TREATMENT

There are three established procedures to be considered in the surgical treatment of pan-

TABLE II.—FINDINGS IN THE BILIARY TRACT IN 9 CASES OF PANCREATIC CYST

Case	Biliary tract history				Operative findings of biliary tract
	Cholecystitis	Gallstones	Jaundice	Previous operations	
1	o	o	o	o	Normal
2	+	+	+	o	Gall bladder scarred and small; 2 gall stones
3	o	o	o	o	Pancreatitis?
4	+	+	+	Cholecystostomy	Several small gall stones. Common bile duct dilated. No stones in common bile duct.
5	+	o	+	Exploratory laparotomy	Normal
6	o	o	o	o	Normal
7	o	o	o	o	Normal
8	o	o	o	Exploratory laparotomy	Normal
9	o	o	o	Exploratory laparotomy	Normal

creatic cysts—excision, external drainage (marsupialization), and internal drainage (anastomosis of cyst to stomach, duodenum, jejunum, or gall bladder).

The ideal procedure is complete extirpation. This is, unfortunately, not always possible because of the intimate relationship of the cyst with the surrounding structures. Other dangers which sometimes make extirpation hazardous are hemorrhage, the possibility of producing diabetes mellitus, or of causing intolerable injury to the intestinal blood supply.

External drainage by marsupialization has been the generally accepted method of surgical treatment up to the present time. It has not, however, been entirely satisfactory owing to the formation of an external fistula which may drain for a prolonged period. Kerr reported a patient having a persistent discharge for 15 years. Judd, Mattson, and Mahorner (13), in their series of cases, reported drainage persisting from a few weeks to 2 years. Others have had the same experience following marsupialization. Other complications also may arise, such as excoriation of the skin by the drainage, infection of the cyst and sinus, and formation of a secondary abscess. Table III shows the duration of drainage in our series.

Many methods have been attempted to eradicate the fistula. Culler used roentgen irradiation, while Hamilton used radium. Others have treated the direct source and attempted to destroy the lining membrane of the pancreatic cyst either mechanically or chemically. Schwyzer applied tincture of iodine to the interior of the cyst; Gordin used a sclerosing agent to destroy the membrane of the cyst.

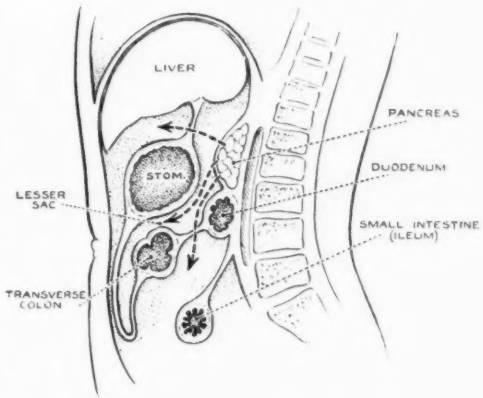

Fig. 1. Sagittal section through the body of pancreas. (Modified from Gray's *Anatomy*, p. 1145.)

To overcome this handicap, a method of internal drainage was devised whereby the pancreatic cyst is anastomosed either to the stomach, duodenum, jejunum, or gall bladder. Jedlicka was the first to attempt an internal anastomosis. He resected a major portion of the cyst and implanted the remainder of the cyst into the wall of the stomach. Others have followed him by implanting a fistula into the stomach or doing a cystogastrostomy. Kerschner used the duodenum in performing his anastomosis. Hahn, in 1927, and Chesterman in 1941, anastomosed the cyst to the jejunum

TABLE III.—DURATION OF DRAINAGE

Case	Type of cyst	Surgical procedure	Duration of drainage	Complications
1	Papillary cystadenocarcinoma	Marsupialization	6 weeks	Died of metastasis 5 months later
2	Pseudocyst	Marsupialization	8 months	None
3	Hemorrhagic	Excision	4 weeks	Mild diabetes mellitus
4	Pseudocyst	Excision	10 weeks	None
5	Pseudocyst	Cystojejunostomy	None	Mild diabetes mellitus preoperatively
6	Papillary cystadenocarcinoma	Cystojejunostomy	None	Patient is symptom-free at present, 6 months after operation
7	Pseudocyst	Incision and drainage	10 days	None
8	Pseudocyst	Implantation of fistula into jejunum	None	Subacute pancreatitis 5 years later
9	Pseudocyst	Implantation of fistula into jejunum	None	Left hypochondriac pain 18 months later

Some surgeons have objected to an anastomosis between the pancreatic cyst and the gastrointestinal tract in the belief that the intestinal contents may enter and lead either to suppuration or to an activation of the enzymes in the true pancreatic cysts. They depended on the resorptive power of the gall bladder to remove the fluid produced by the cyst. Walzel performed a primary anastomosis between the cyst and the gall bladder and, to prevent a reflux of bile, the cystic duct was ligated. The rationale for the cystic duct ligation is difficult to understand. Neuffer performed a similar operation but did not ligate the cystic duct. Meyer obtained satisfactory results using the technique of Neuffer.

SURGICAL PROCEDURE

In 2 cases encountered in the clinic in the past 2 years, the patients were treated by doing a cystojejunostomy. The results to date have been very satisfactory and the surgical procedure is described below.

Exposure of the pancreatic cyst is made either through the gastrocolic or the gastrohepatic omentum. A rent is made in the mesocolon, through which a loop of jejunum about 35 centimeters from the ligament of Treitz is drawn. The jejunal serosa is sutured to the cyst wall with a row of interrupted silk sutures. The cyst is then aspirated and an incision 2.5 centimeters in length is made in its wall. A similar parallel incision is made in the jejunum. A posterior layer of sutures unites the cyst lining with the intestinal mucosa. A row of interrupted Connell sutures is placed, thus inverting the anterior walls of the cyst and jejunum. This is reinforced with another layer of interrupted silk sutures. Two sutures are placed at each angle to buttress the jejunum to the pancreas. A jejunojejunostomy of the efferent and afferent loops is then made about 15 centimeters from the ligament of Treitz.

CASE REPORTS

CASE 1. A white female, aged 43, was seen at the clinic on July 19, 1927, because of soreness and pain over the right upper abdomen, which had been present since April 9, 1927. This had persisted for several days and was not related to food intake. On May 9, 1927, she began to have sharp pain over the same area, accompanied by nausea and vomiting, and morphia was required for relief. On June 3, 1927, a mass appeared in the left upper quadrant.

Laboratory studies were negative. Fluoroscopic examination showed an extrinsic pressure defect on the stomach, between the antrum and media, causing an hour-glass deformity. The interpretation was pancreatic tumor.

On July 22, 1927, operation was performed by Dr. F. H. Lahey. The approach was made through the gastrohepatic omentum. A cyst about 7 centimeters in diameter was found projecting from the body of the pancreas. The cyst was opened, and the friable contents removed. The lining was painted with iodine, and a cigaret drain inserted. The edges of the cavity were sutured to the peritoneal edges around the drain.

The pathologic report was papillary cystadenocarcinoma of the pancreas.

There was no postoperative complication. The patient was discharged on the tenth day with a discharging sinus.

Follow-up examination of this patient showed a small discharging sinus on August 7, 1927. On September 11, 1927, there was no sign of diabetes mellitus. The patient died December 11, 1927, from metastasis of this carcinoma.

CASE 2. A white female, aged 71, came to the clinic on September 25, 1941, because of an abdominal mass of 3 years' duration. She complained of occasional dull pain in the right upper quadrant of the abdomen which occurred after eating. She had had a short period of jaundice about 4 years previously. In 1932 she was operated upon for pancreatitis. The findings at this operation are not available.

Physical examination showed a tumor mass filling the upper abdomen, extending about 5 centimeters below the umbilicus.

Laboratory studies were negative. A partial gastrointestinal roentgenologic examination showed the stomach to be displaced by an extragastric mass lying along the lesser curvature. An intravenous pyelogram showed displacement of the left ureter by this mass, with probable pressure on the left renal pelvis.

Operation was performed September 29, 1941 by Dr. S. F. Marshall. A cyst about 25 centimeters in diameter was found lying posterior to the lower portion of the stomach and one of the loops of the small bowel. Four liters of fluid was removed from the cyst. A stab wound was made in the left flank. The wall of the cyst was brought out through this incision and sutured to the peritoneum and muscle. It was likewise sutured to the skin with interrupted black silk sutures. Two cigaret drains were placed into the interior of the cyst. The gall bladder was small and scarred, and contained two stones.

The patient was discharged on October 19, 1941, and was seen in the clinic thereafter. The drainage continued until May 21, 1942. The patient was seen on July 16, 1943; there was no recurrence of the cyst

and drainage had ceased. There was no sign of diabetes mellitus. On October 17, 1943, she died from a cerebrovascular accident.

CASE 3. A white female, aged 58, was seen in the clinic on May 18, 1943. For the past 5 years she had suffered from intermittent attacks of nausea. A roentgenogram taken by her physician was said to show a cyst of the liver. One week previously sharp pain occurred under the left scapula; the pain extended to the right side.

On physical examination, an irregular mass, which moved with respiration, was outlined just below the left costal margin.

All laboratory studies were negative.

An exploratory laparotomy was performed on June 14, 1943, by Dr. S. F. Marshall. A cyst about 10 centimeters in diameter was found extending from the body of the pancreas. It was located posterior to the stomach and extended upward against the gastrohepatic omentum. A smaller cyst, 2 centimeters in diameter, was located posteriorly and attached to the posterior abdominal wall. These two cysts were excised. Two cigaret drains were placed in the area through a stab wound in the left flank.

The pathologic report was hemorrhagic cyst with fibrous wall, probably the result of localized pancreatitis.

Follow-up examination on August 6, 1943, demonstrated mild diabetes. On November 29, 1943, a glucose tolerance test showed mild diabetes mellitus. Drainage had ceased on August 6, 1943.

CASE 4. A white female, aged 39, was seen at the clinic on November 15, 1943. About 2 years previously, cholecystostomy was performed elsewhere for attacks of right upper quadrant pain and vomiting. Many stones were removed from the gall bladder at that time. Mild jaundice was present before that operation. Since then she had had one attack of pain over the right upper quadrant, which lasted for 2 weeks. There was no jaundice.

Physical examination gave negative results. All laboratory studies were negative. A cholecystogram showed an outline of the gall bladder containing several very small negative shadows. Roentgenograms taken after a barium meal and barium enema were normal.

On January 14, 1944, operation was performed, by Dr. C. C. Engleman. A cyst, measuring about 2 centimeters in diameter, was found at the root of the mesocolon. Several small gallstones were found in the gall bladder. The common bile duct was dilated and contained no stones. The cyst was located at the inferior border of the head of the pancreas. The pancreas was hard, and the capsule thickened, suggesting a previous attack of pancreatitis. The cyst was excised. Cholecystectomy and choledochostomy were carried out. A cigaret drain was placed in the pancreatic area, and a Penrose drain placed in Morrison's pouch.

The pathologic report was cyst, with fibrous wall.

Follow-up examination on February 16, 1944, showed no drainage from the site of the pancreatic cyst. The patient has had no recurrence of symptoms or cysts.

CASE 5. A white male, aged 45, was seen at the clinic on February 2, 1944. In 1939, laparotomy had been performed elsewhere for cholecystitis. The gall bladder was not removed. However, an appendectomy was done. Following this he had had abdominal pain intermittently. Three weeks previously he had had an attack of jaundice which lasted for 3 days. He had lost 30 pounds in weight in 2 months.

Physical examination gave negative results. All laboratory studies were negative except for a glucose tolerance test which was suggestive of diabetes mellitus. A cholecystogram was negative. A roentgenogram taken after a barium meal showed an enlargement of the duodenal loop with compression of the first portion of the duodenum.

Operation was performed on March 1, 1944, by Dr. Ralph Adams. A cyst about 6 centimeters in diameter was found arising from the head of the pancreas and occupying a position in the curve of the duodenum. This cyst was anastomosed to the jejunum as described previously.

The pathologic report was fibrosis and chronic inflammation.

The patient was discharged on March 18, 1944, after an uneventful convalescence. He was seen on April 28, 1944, and at that time he had no symptoms of any distress. On May 5, 1944, a gastrointestinal roentgenologic study was done. This showed a decrease in the size of the cyst, with no evidence of any barium in the cyst cavity. On August 15, 1945, another fluoroscopic and gastrointestinal roentgenogram again showed a decrease in the size of the cyst; no barium entered the cyst.

CASE 6. A white female, aged 51, was seen at the clinic on May 22, 1945, complaining of epigastric discomfort for the past 3 years. The gnawing ache in her epigastrium was aggravated by eating. About 6 months previously she noticed a mass in the left upper quadrant which steadily increased in size.

Physical examination showed a mobile smooth mass in the left upper quadrant. All laboratory studies were negative. Fluoroscopy and gastrointestinal roentgenograms showed a deformity of the stomach produced by a large mass in the left upper quadrant. An intravenous pyelogram was normal.

Operation was performed on June 23, 1945, by Dr. Ralph Adams. A large cyst, 12 centimeters in diameter, was found arising from the body of the pancreas. The stomach was displaced downward. The cyst contained many papillomatous masses, and frozen section showed it to be carcinomatous. Cystojejunostomy was performed as described previously.

The pathologic report was papillary cystadenocarcinoma.

The patient made an uneventful convalescence and was discharged on July 15, 1945. She was seen in the clinic on August 15, 1945, and had no complaints. The mass was still palpable. Fluoroscopy and gastrointestinal roentgenograms showed the cyst with no barium entering it.

CASE 7. A white female, aged 41, was seen at the clinic on June 29, 1945, complaining of weakness, dizziness, and attacks of unconsciousness. Studies showed her to have an islet cell tumor of the pancreas. On July 5, 1945, an operation was performed, and an islet cell tumor in the head of the pancreas was removed. The pathologic report was islet cell adenocarcinoma.

On September 10, 1945, pain appeared in the left hypochondrium. This pain was fairly severe and extended to the back. Roentgenographic examination of the stomach showed a crater deformity on the posterior wall of the fundus of the stomach.

On October 4, 1945, an exploratory laparotomy was performed by Dr. S. F. Marshall. A collection of slightly cloudy fluid in a thin walled cyst was found in the left subphrenic area, and also between the stomach and pancreas. The pseudocyst was evacuated. The wound healed on removal of the drain, and she was discharged on the nineteenth postoperative day, symptom-free. No evidence of metastasis or recurrence of adenocarcinoma of the pancreas was found.

CASE 8. A white male, aged 18, was seen at the clinic on January 22, 1936, complaining of a draining abdominal sinus for the past 3 months. About 3 months previously following an upper abdominal injury, an exploratory laparotomy was performed elsewhere, and a large hematoma was found lying between the two layers of the gastrocolic omentum. The abdomen was closed without drainage. The postoperative course was complicated by the appearance of a discharging sinus.

Physical examination showed a discharging fistula in a healed upper right rectus incision, surrounded by an area of irritated skin.

On January 22, 1936, a gastrointestinal roentgenogram, taken after lipiodol injection of the sinus, showed the fistula to be in the region of the head of the pancreas.

Conservative management was then instituted with diet, alkaline powders, and belladonna. When no improvement was shown under this regimen, the pancreatic fistula was transplanted into the jejunum on March 16, 1936 (operation performed by Dr. F. H. Lahey). The postoperative course was uneventful, and the patient was discharged from the hospital on April 3, 1936.

In August 1936, another abdominal injury necessitated another exploratory laparotomy elsewhere. The fistulous tract into the jejunum was not injured.

The patient was symptom-free until July 22, 1941, when severe pain developed in the upper left portion of the abdomen, accompanied by nausea, vomiting, and a temperature of 102 degrees F. He was then admitted for study. A diagnosis of subacute pancreatitis was made. Following a period of medical management, he was discharged on October 10, 1941, improved.

CASE 9. A white male, aged 32, was seen at the clinic on June 18, 1944, complaining of a draining pancreatic fistula of 15 months' duration. In May 1942, an exploratory laparotomy was performed elsewhere for acute pancreatitis. No drainage was carried out. In March 1943, this patient was operated on elsewhere, presumably to have a cholecystectomy performed for a nonfunctioning gall bladder. A pancreatic cyst was found, however, and since it was too small to be marsupialized, it was opened and drained with a cigaret drain. This drain was removed 3 months later. Since then the sinus has continued to drain, occasionally requiring probing to relieve the obstruction which caused a great deal of pain.

Physical examination showed a healed upper right rectus incision with a small fistulous opening at the upper end of the scar. The surrounding skin showed a chronic irritation. All laboratory studies were negative.

On June 20, 1944, the fistula was transplanted into the jejunum, Dr. F. H. Lahey performing the operation. The postoperative course was normal, and the patient was discharged on July 19, 1944.

Follow-up by correspondence showed this patient to be symptom-free until December 19, 1945, when he revealed that he has been having left hypochondriac pain extending to the back for the past month. He is to be readmitted for further study.

SUMMARY

The literature on pancreatic cyst is reviewed. Nine additional cases are reported. The anatomic, physiologic, and pathologic features peculiar to this lesion are enumerated. The symptoms, signs, and examinations upon which a diagnosis may be made are discussed. Internal drainage by means of cystojejunostomy is recommended when extirpation of the cyst is not feasible, and the technique employed in establishing this anastomosis is described.

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SEVERE BURNS: CLINICAL FINDINGS WITH A SIMPLIFIED PLAN OF EARLY TREATMENT

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DESPITE the tremendous amount of clinical and experimental study, much of it stimulated by the war, there is considerable confusion in the minds of many surgeons as to the preferred method of immediate and early treatment when confronted by a serious thermal injury. The purpose of the present communication is to describe the clinical findings following a plan of treatment reduced to its simplest details. It must be emphasized, however, that no plan can take the place of careful clinical study of the individual case. Nevertheless, the results obtained seem sufficiently good to suggest that therapy based on such a plan may be preferable to more intricate and complicated methods. The large literature on the treatment of burns will not be reviewed; it has been well done by Harkins and others.

SELECTION OF MATERIAL

During the 2 year period of study 112 burn cases were considered severe enough to justify admission of the patient to the hospital. Only about half, or 55 cases, were selected for analysis. First, no case was included in which the patient did not arrive in the hospital within a few hours of injury and in which he did not remain in the hospital until full treatment was carried out. Second, in order to study only severe cases, all patients with burns involving less than 10 per cent of the body surface were excluded, with the exception of 4 patients in this group requiring skin graft.

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PLAN OF THERAPY

On entrance into the receiving room all exposed burned areas were covered with a sterile sheet and blanket. Morphine or codeine injections were not given as routine but only to control pain or apprehension, respectively, when present. As routine a prophylactic dose of antitetanus serum was injected. Full treatment was carried out in the following chronological order after arrival at a dressing room off the surgical wards.

Intravenous injections. It was accepted as axiomatic that the voluntary oral route for the administration of needed food and fluid elements was preferable to the parenteral channel, which should be employed only when necessary. In other words, only when the oral route was ineffective, particularly in meeting acute deficits, were intravenous injections used. Plasma transfusions were therefore not given as routine, were not based on the extent of the burn, but were used primarily for the treatment of shock when present, or prophylactically when shock seemed imminent. This routine required constant observation of the patient, particularly during the first 24 to 48 hours, inasmuch as some patients entered the hospital in good general condition, but developed signs of shock after several hours, or responded to one transfusion only to fall into shock again later. Although pronounced hemoconcentration is

often used as an indication for the injection of plasma, and did nearly always accompany the appearance of shock, it was used as a routine indication in the present series only when the red count increased progressively. Many patients with hemoconcentration were not in shock and recovered without any intravenous therapy. This fact was also true in several cases with a large involvement of the body surface. If there were no systemic manifestations other than moderate hemoconcentration, plasma was, with few exceptions, withheld. Whole blood was given only when evidence of anemia developed or in conjunction with skin grafting. Saline and glucose solutions were injected only in connection with plasma transfusions and in the only case with persistent vomiting. A high fluid intake by mouth was maintained from the beginning, as described in detail under nutrition.

The amount of plasma given depended on the severity of the shock. No rigid formula was used, but for an adult at least 500 cubic centimeters was started as the initial dose. In the most severe cases larger amounts, e.g., 20 cubic centimeters per kilogram of body weight, or about 1500 cubic centimeters for an average sized adult, were injected. The rate of administration was quite rapid to combat shock, and more plasma was injected if the clinical response was not sufficient, or was repeated if circulatory impairment recurred.

Débridement and cleansing. No débridement whatever was carried out in any patient even in several in whom home remedies had been applied, such as talcum powder, sulphur ointment, lard, baking soda, bluing, and molasses. However, none of the burns was grossly dirty. Blisters were left intact and no attempt whatever was made to remove shreds of skin or other loose tissue. Cleansing was gently carried out with soap and water, but largely over the intact skin next to the burned area. Omission of débridement in fresh, clean burns was first formally recommended by Cope as a result of treating the victims of the Cocoanut Grove disaster; its use in the present plan was based largely on his excellent results.

Dressing. The basic principles involved in the application of a dressing to the burned area in order to avoid infection was thoroughly described by Colebrook and his coworkers. Their excellent results prompted the use of these principles. The first and most important principle was the maintenance of scrupulous aseptic technique, the same, indeed, as that employed in the operating room. A rigidly observed detail was the masking of the patient and of all personnel during dressing. Next to the burn a layer of fine mesh gauze or other fabric was applied with or without some type of ointment. Over the primary layer of fabric a bulky layer of gauze was applied and held in place with a firm circular bandage. Pressure by means of cotton waste or elastic bandage was used in a number of cases, but was not adopted as a routine. A plaster splint for the extremities was sometimes included in the dressing. A bulky dressing, however, was always used because it absorbed discharges and seemed partly to immobilize the part.

Another important detail was the application of dressing well beyond the obviously burned area of skin, for reasons mentioned below. A third feature of the dressing was the fact that it was allowed to remain in place as long as possible, usually 10 to 14 days. It was removed earlier only when necessary because of pain, high fever, or excessive discharge. It was found advisable to explain this policy of infrequent dressing to the patient and the family in order to prevent a tendency for them to complain of neglect, which frequently occurred unless the plan was explained beforehand.

Burns about the buttock. In burns in the general region of the anus, particularly in children, a constipating regimen with paregoric was instituted and maintained for about a week. After this period enemas were ordered and given each day at a specific time when a member of the house staff was present, so that the dressing could be changed at once if necessary in order to prevent undue contamination. This seemed to work very well and probably played an important part in the prevention of secondary infection.

Oxygen inhalations. Patients who showed evidence of pulmonary injury or those suffering from respiratory distress for other reasons were placed in an oxygen tent during the first few days.

Chemotherapy. No local chemotherapy was used whatever. As a routine, sulfathiazole, 1/10 gram per kilogram per day was given by mouth. However, if there was any contraindication to the use of sulfathiazole, it was not given. In a few patients penicillin was used early in doses of 160,000 units per day, but more commonly and effectively later in preparation for skin grafting. All primary chemotherapy was discontinued by the end of the first week unless there was evidence of infection.

Preparation for skin graft. Preparation for skin graft presented the greatest problem in the treatment of full thickness burns and will not be described in this paper. All sorts of methods were used, including proteolytic ferments. In general, wet saline packs were found to be the most effective, although they left much to be desired.

Nutrition. Though listed last, attention to nutrition, including oral fluid intake, was given a high priority in the present plan of treatment. Practically all patients suffered great thirst on admission and it was not necessary to urge them to ingest a large quantity of liquids. From the very beginning these liquids were of high nutritional value and included high protein and caloric drinks, ice cream, etc. A high intake of sodium chloride based on the experimental findings of Rosenthal and the clinical studies of Fox was not a part of the present plan. It is likely, however, that all patients did ingest large amounts of electrolyte in their food and drink. Vomiting was infrequent, but when it occurred was usually transient or beneficially influenced by plasma transfusions, in patients suffering from circulatory impairment and in whom vomiting was a manifestation thereof.

The assurance of a good nutritional intake was not left to chance, to a dietary order, or to the efforts of the dietician. A specific amount of fluids and food was ordered just as in the case of medication. Moreover, the intake was checked each day to be sure that most of it was taken. In addition, the urinary output was measured and if it fell below 1000 cubic centimeters the intake was checked, and if inadequate, efforts were made to see that it was maintained at several liters or more a day. If the patient refused to eat a normal, well balanced diet, a high protein milk diet was ordered which in one liter contains 100 grams of protein and 1000 calories, which was considered as the daily minimum for an average sized adult. When possible the patient was urged to take 200 grams of protein and 2000 or more calories, particularly after the first few days. The threat of tube feeding was used in many patients, but was not employed in any of the present cases. Large amounts of ascorbic acid were given, 1 gram or more a day. An important feature of a good nutritional intake is that it start at once, i.e., as soon as the patient is dressed. Prevention is far easier than cure. If severely burned patients are allowed to lose much body tissue, the problem of restoration is extremely difficult even with a large dietary intake. The importance of maintaining good nutrition in burned patients has been well and extensively studied by Levenson, Davidson, Lund, and Taylor.

Early movement of the patient was encouraged and, unless the lower extremities were involved, the patient was urged to get up out of bed as soon after the dressings were applied as possible. If the lower extremities were involved, an attempt was made to get the patient into a wheel chair. When the upper extremities were involved it was obviously necessary to provide frequent nursing care in order to insure an adequate nutritional intake.

CLINICAL FINDINGS

Because of the tremendous variation in the findings it is difficult to summarize them adequately. To aid in the analysis, all patients were divided into three groups and the main details summarized in three tables as follows: Table I, those which healed without the use of skin graft; Table II, those which healed with skin graft; Table III, fatal cases.

Text figures of changes in the red count will be presented to illustrate findings of special significance. Reference will be made to them in the following general discussion of the results of the study.

Systemic manifestations on entrance. The most common symptom was thirst, which was almost universally observed in practically all

TABLE I.—BURNS WHICH HEALED WITHOUT SKIN GRAFT*

Case	Age, sex	Extent (% body surface)	Fever(°F.) Duration (days)	Day 1st change of dressing	T(°F.) day 1st change of dressing	Day of discharge	Thermal agent	Hemoconcentration Highest RBC (mill/c. mm.)	Remarks
1	25-F	30	None	12	Normal	37	Flame	6.1	500 c.c. plasma
2	4-F	15	None	13	Normal	25	Hot water	5.1	
3	8-F	15	101-1	13	100	31	Hot water	5.3	
4	1-M	20	100-2	14	Normal	26	Hot water	5.25	
5	1-M	40	102-1	13	Normal	36	Hot water	5.62	
6	3-M	30	102-1	6	101	39	Hot water	8.2	See Fig. 1
7	6-F	19	None	13	Normal	15	Hot water	None	
8	2-M	20	104-1	6	101	18	Hot water	5.2	
9	9-M	10	None	14	Normal	14	Flame	None	
10	49-F	40	None	12	Normal	28	Flame	6.8	See text Fig. 2
11	32-F	25	101-1	13	Normal	84	Flame	5.8	500 c.c. plasma
12	14-F	10	None	6	Normal	8	Hot water	None	
13	21-F	20	None	12	Normal	12	Hot water	6.5	
14	25-F	13	None	7	Normal	12	Flash	None	
15	3-M	12	102-2	12	Normal	28	Hot water	None	
16	39-F	15	None	9	Normal	13	Hot water	None	
17	1-F	30	102-f	8	101	17	Hot water	None	
18	51-M	45	100-4	5	Normal	59	Hot water	5.5	See text Fig. 3 1600 c.c. plasma
19	5-F	10	None	5	Normal	18	Hot water	None	
20	7-M	30	None	10	Normal	63	Hot water	None	
21	5-F	40	100-2	13	Normal	22	Hot water	None	
22	1-M	15	None	5	Normal		Hot water	None	
23	10-M	20	None	9	Normal	13	Flame	None	
24	1½-M	15	None	10	Normal	15	Hot water	None	
25	1½-M	10	None	19	Normal	20	Hot water	None	
26	70-F	30	103-3	8	101		Flame	5.1	

*None showed systemic manifestations on admission.

of the patients. Second in frequency was a complaint of coldness and chilliness, which practically always disappeared as soon as the burned area was covered by sterile sheets and blankets and permanently controlled by the application of the dressing. Pain was strikingly rare, which is of extreme interest in view of the almost universal use of morphine in the treatment of burns. Indeed, many of the patients were rather apathetic and had no complaints. In only 9 of the 55 cases herein reported was morphine used, and in many of these merely to control restlessness rather than pain. In an unpublished series of observations on human volunteers this finding was confirmed, i.e., the intense pain stimulus at the time of the burn was replaced by a dull ache which lasted for a short time and then gradually disappeared. It may be that certain other types of burns not observed in the present series are accompanied by lasting severe pain, but this certainly was not true of the patients we observed. The possible dangers and misuse of morphine in severe injuries has been well discussed by Beecher.

Vomiting occurred in only 2 of the nonfatal cases and in neither of them did the vomiting interfere with feeding because it disappeared within 24 hours. In the fatal cases vomiting was more frequent; 6 of the 12 patients vomited, but in only 1 was it persistent (Case 51). Evidence of shock and toxemia was

TABLE II.—BURNS WHICH HEALED WITH SKIN GRAFT

Case	Age, sex	Extent (% body surface)	Fever(°F.) Duration (days)	Day 1st change of dressing	T(°F.) Day 1st change of dressing	Day of discharge	Thermal agent	Hemoconcentration highest RBC (mill./c. mm.)	Remarks
27	15-M	15	104-5	12	Normal	240	Flash	None	
28	3-M	10	102-1	12	Normal	150	Flame	5.8	500 c.c. plasma
29	38-F	30	102-5	11	101	150	Flame	4.2*	500 c.c. plasma
30	21-F	40	104-2	12	100	90	Flame	8.9	1000 c.c. plasma (see text Fig. 4)
31	16-M	20	103-2	12	100	90	Flash	5.2	500 c.c. plasma
32	6-M	6	None	10	Normal	34	Flame	None	
33	24-F	30	101-2	10	100	138	Flash	5.8	1500 c.c. plasma (see text Fig. 5)
34	25-F	10	None	10	Normal	44	Flame	None	
35	9-M	12	101-3	9	100	124	Flash	None	
36	1½-M	5	103-5	6	100	59	Hot water	None	
37	45-M	6	102-1	4	100	120	Flame	None	
38	4-M	6	None	10	Normal	102	Flame	None	
39	3-M	38	None	10	Normal	104	Hot water	None	
40	7-M	30	104-1	13	101	—	Flash	8.0‡	1000 c.c. plasma (see text Fig. 6)
41	½-F	40	104-1	7	102	120	Flame	5.0‡	250 c.c. plasma
42	28-F	30	103-1	8	101	340	Flame	None	7 mo. pregnant Aborted 24 hrs.
43	2-F	12	101-2	5	101	—	Flame	None	

*Patient malnourished and probably anemic at time of burn.
‡Only patients of this group in shock or with toxemia soon after admission to hospital.

absent in all of the patients summarized in Table I, all but 5 of whom suffered from hot water burns, even though the extent of the burn in this group was as great as 40 per cent of the body surface. Even in the group of patients requiring skin graft (Table II) evidence of shock and toxemia was present in only 2 cases. On the other hand, these signs were present in 8 of the 12 fatal cases (Table III). The term shock was applied to any patient suffering obvious circulatory impairment. The term toxemia was used when stupor, irrational behavior, restlessness, delirium, and high fever were observed. In a few cases there was moderate transient lethargy, but this was not listed as a systemic manifestation.

Hemoconcentration was studied in practically all cases by taking frequent red cell counts or hematocrit measurements. Great variations and differences were detected in the behavior of the hemoconcentration, 6 examples of which are shown in the accompanying text figures. It will be noted by consulting these and the tables that flame and hot water burns exhibit differences in both the frequency and duration of hemoconcentration. In general, fewer flame burns showed increases, but the process seemed more prolonged yet less intense than in hot water burns.

Not listed was the relationship between the character of the necrotic skin and the presence and severity of systemic manifestations, including fever. In general, moist necrosis was much more likely to present signs of toxemia whereas a dry, hard necrosis was associated with fewer systemic signs (8). This was particularly true after the first 24 to 48 hours.

Estimation of the degree and depth of the burn. The area of the skin actually burned was nearly always greater than that estimated by inspection at the time of entrance. This was true especially of hot water, but also of flame and flash burns. This finding was of obvious practical importance because it emphasized the need for applying the dressing well beyond the visible burned skin. Otherwise, exposed injured skin would become apparent within 2

TABLE III.—BURNS WHICH ENDED FATALLY

Case	Age, sex	Extent* (% body surface)	Systemic manifestations on admission	Highest T (°F.)	Time of death (after admission)	Thermal agent	Hemoconcentration Highest RBC (mill/c. mm.)	Remarks
44	35-F	36	None	103	108 days	Flame	5.8	Pt. had multiple sclerosis. Died after transfusion reaction (Rh?). 3000 c.c. plasma, also whole blood and red cells
45	70-M	62	None	102	7 days	Flame	7.8	1500 c.c. plasma
46	17-M	56	None	102	30 days	Flash	6.3	1000 c.c. plasma
47	43-M	90	Shock Toxemia	99	4 days	Flame	7.7	1000 c.c. plasma
48	57-M	100	Shock	97	3 hours	Flame	5.3	500 c.c. plasma
49	2-F	100	Shock	97	5 hours	Flash	?	500 c.c. plasma
50	2-M	20	Shock	99	6 hours	Flame	6.95	
51	3-F	20	Shock	100	24 hours	Flame	6.1	Died during plasma transfusion
52	42-M	75	Toxemia	100	12 days	Flame	8.25	6000 c.c. plasma
53	2-F	30	Toxemia	99	2 days	Flame	5.2	500 c.c. plasma
54	53-M	40	None	103	14 days	Flame	None	500 c.c. plasma 1000 c.c. whole blood
55	4-F	75	Shock Toxemia	101	6 hours	Flame	7.3	750 c.c. plasma

*Estimate only approximate in patients dying soon after admission.

or 3 days, invariably become infected, and act as a portal of entry for bacterial invasion.

The depth of the burn was also difficult to predict by inspection alone even at the time of the first dressing. By experience, however, it was soon obvious that the kind of thermal agent was of primary importance. For example, hot water seldom produced full thickness burns, only 2 out of 21 in this category requiring skin graft. Moreover, if one consults Table I, listing the burns which required no skin grafting, 19 of the 26 were due to hot water. On the other hand, in Table II, listing those which required skin graft, in all but 2 of the 17 cases a flame or flash was the responsible thermal agent. It should be emphasized, however, that 7 of the flame burns healed without skin graft, even though in 4 of them the burn had involved 25 per cent or more of the body surface (Cases 1, 10, 11, 26). It was also observed that even in extensive flame burns requiring skin graft the area was frequently smaller than expected. This seemed directly connected with the absence of infection and with the fact that the patient maintained a good nutritional intake from the beginning. Entirely apart from the food intake during treatment, the nutritional status of the patient at the time of injury also played an important part in the extent of skin requiring graft. Many of the burned patients were obviously malnourished and in them the depth of the burn always seemed to be much greater than in healthy individuals.

Type of ointment. Observations made with various types of greasy ointments showed in general relatively little difference even when similar areas were compared. However the best results seemed to follow the use of a water soluble carbowax ointment made up according to directions supplied by Dr. F. L. Meleney. Comparisons were also made between petrolatum-impregnated gauze and dry fine mesh gauze. In general, the latter, placed next to the skin, was followed by a drier wound but removal was more difficult and painful. Petrolatum, if applied sparingly in fine mesh gauze, proved fairly satisfactory; however, if an excessive amount of it were applied, which was more likely if coarse mesh gauze were impregnated with it, there was much more maceration of tissue and evidence of irritant action, as shown by greater discharge and apparently more necrosis.

Incidence of infection. The most striking observations made in the present series of cases was the relatively low incidence of infection. This was obvious at the time of the first change of dressing even when a period of 2 weeks had elapsed. In many cases the burned areas were dry and healed. Even when the skin showed moist necrosis there was rarely any pus or cellulitis. This low incidence of infection is also shown by the relatively low incidence of fever, which is shown in the tables herewith presented. For example, 19 of the 43 healed cases showed no fever at any time and 29 no elevation above 101 degrees F. More striking was the fact that at the time of the first change of dressing only one patient had a fever of 102 degrees F. or above, only 8 had 101 degrees F., 7 had 100 degrees F.; in 27 of the 43 the temperature was normal.

Another of the advantages of infrequent dressings was a reduction in the amount of routine nursing and professional care. This made it possible to employ the available nursing service for other purposes, notably maintenance of a good fluid and food intake, psychotherapy, and early movement and ambulation of these patients.

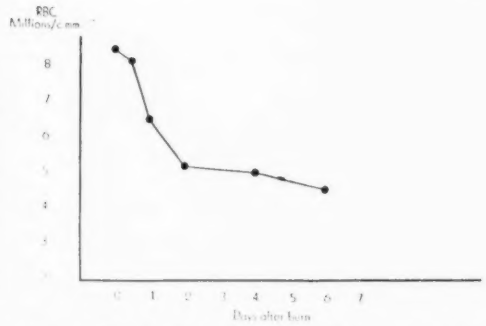

Fig. 1. Case 6. Three year old male, hot water burn involving 30 per cent of the body surface, which healed without skin graft. Note pronounced hemoconcentration on entry and its prompt subsidence without parenteral fluids. There were no systemic manifestations at any time except moderate lethargy for the first 24 hours. Fluid and food intake excellent from the beginning.

Parenteral injections. Only 10 of the 43 patients with healed burns were given plasma or blood transfusions as part of their early therapy, and of these 9 had flash or flame burns. Many extensive burns healed completely and satisfactorily without the use of plasma. This was true particularly of hot water burns, even when the red count was

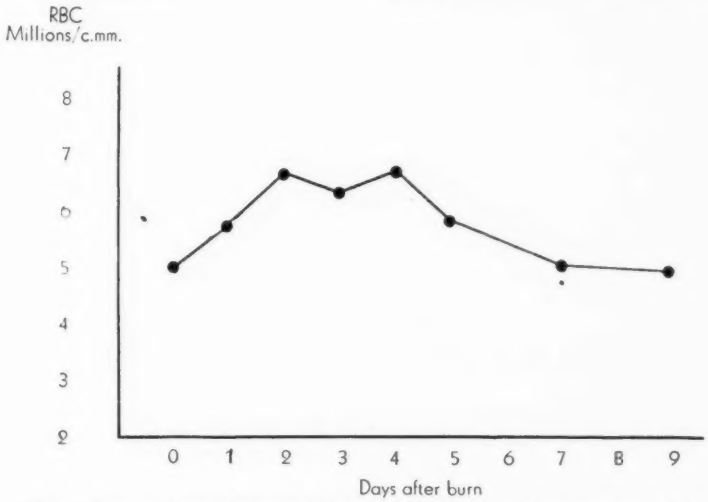

Fig. 2. Case 10. Forty-nine year old female, flame burn involving 40 per cent of the body surface, which nevertheless healed without skin grafting. Note the slow appearance of moderate hemoconcentration, which reached its maximum only after 48 hours and disappeared slowly by the 7th day. No parenteral fluids were used, but the patient took a high caloric, high protein diet from the beginning.

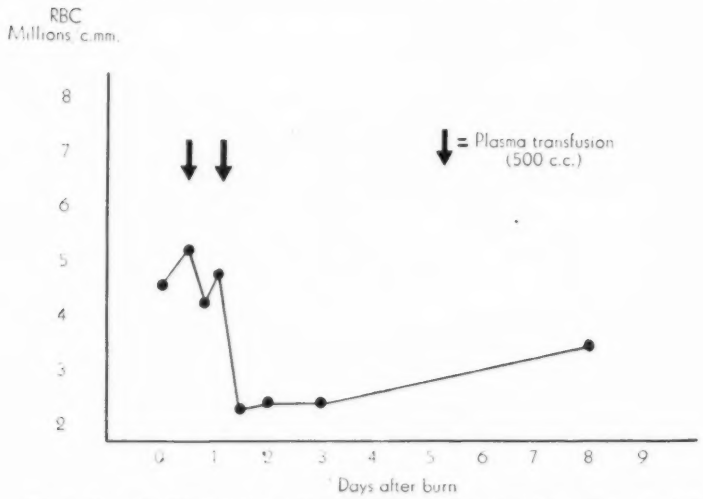

Fig. 3. Case 18. Fifty-one year old male, hot water burn involving 45 per cent of the body surface, which healed without skin graft. Note the prompt correction and probable overcorrection of hemoconcentration following two plasma transfusions. The anemia showed some spontaneous correction but was subsequently treated with whole blood transfusions. The general condition was good throughout the hospital stay.

significantly, and once greatly elevated (Figs. 1 and 2). The effect of plasma in correcting the hemoconcentration is well demonstrated in several cases shown in these figures. It should be mentioned, however, that 2 of the fatalities (Cases 50 and 51) might have been avoided had plasma been given or repeated, although in both patients pulmonary complications may have contributed to the fatal outcome. It should be noted that when plasma was needed large volumes were usually used. For example, in Case 52, 6 liters were used before the level of the red count remained normal.

Rapidity of healing. As compared with our previous experiences, the patients in the present series, even many with extensive burns, healed more rapidly and required less skin grafting. This seemed undoubtedly the result of the relative decrease in infection; infection itself is known to convert second degree into third degree burns, and thus increase the number requiring skin graft. It was striking that several of the extensive, fairly deep burns healed spontaneously or required only a small skin graft. Perhaps of equal importance in the rapidity of healing, as already mentioned, was the high nutritional intake. On several occasions, patients whose nutrition was particularly well maintained by a high food intake had deep burns which looked as if they would require extensive skin graft, but which healed spontaneously without contracture, or needed only small coverage.

Removal of slough. The results in this group of patients were unsatisfactory and accounted for the long period of hospitalization, as shown in Table II. It is probable that further research will improve the present methods.

Anemia. Anemia was observed in only 8 of the 55 cases. In 6, the anemia was only moderate; the lowest count was between 3 and 4 million. In only 2 cases did the red count fall below 3 million. Three of the 8 cases resulted fatally, and this included one of the patients with a count under 3 million. On the other hand, the patient with the lowest red count, 2.4 (Case 18) recovered uneventfully. Whole blood transfusions as well as packed or suspended red cells were used in all of these cases, but not as an initial form of therapy, inasmuch as anemia was not found during the first 24 hours, and in only 1 case (Case 18, which recovered) as early as the second day.

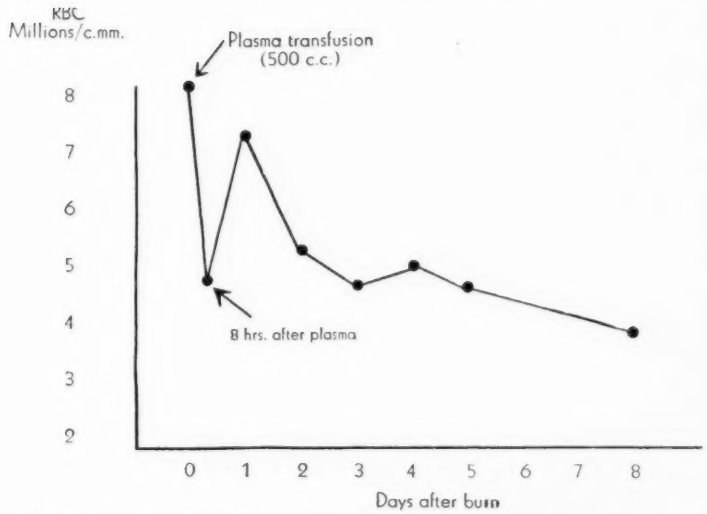

Fig. 4. Case 30. Twenty-one year old female, flame burn involving 40 per cent of the body surface requiring some skin grafting. Note the hemoconcentration on admission which responded promptly to a plasma transfusion only to recur within 24 hours. Spontaneous correction occurred thereafter, undoubtedly aided by an excellent fluid and food intake by mouth.

Blood chemical changes. Plasma protein determinations were made in many of the patients but will not be summarized in this report. Frequent red cell counts or hematocrits were made and form the basis for many of the graphs presented herein. The nonprotein nitrogen of the blood was determined in many cases and frequently was found to be elevated, returning to normal with satisfactory progress of the case. In two of the fatal cases the rise was progressive until death.

Fatal cases. Inasmuch as the saving of life comes first in treatment, failure to do so under the present plan of treatment deserves special analysis. Scrutiny of the 12 fatal cases listed in Table III indicates that 6 of them were flame burns which involved 60 per cent or more of the body surface and might therefore be said to be fairly hopeless from the start. In spite of this, one of these patients survived for 7 days, another for 4 days, and a third for 12 days, the 3 others dying within a few hours after admission. The remaining 6 fatal cases might perhaps be called preventable failures. In 1, the patient died 3 months after the burn following a fatal (Rh?) transfusion reaction. In another (Case 46) the patient survived for 30 days even though the burn involved 56 per cent of the body surface. Progress was satisfactory even though tetanus developed (in spite of the prophylactic injection of serum on admission) and apparently responded well to therapy. While it may have been partly the cause of death, the presence of an old Pott's disease of the spine suggested that miliary tuberculosis may have developed terminally. In another case involving 40 per cent of the body the patient survived 14 days (Case 54). This patient presented great difficulty throughout his hospital stay, largely because of repeated hemorrhage from the burned area. The burn was quite severe, inasmuch as the deep fascia was exposed at numerous places. Bleeding might have been due to hypoprothrombinemia. Unfortunately, no observations thereof were made. Another fatal case involving only 30 per cent of the body occurred in a 2 year old female who lived only 2 days (Case 53). The patient entered somewhat stuporous and this increased until the time of death without any fever. Respirations were

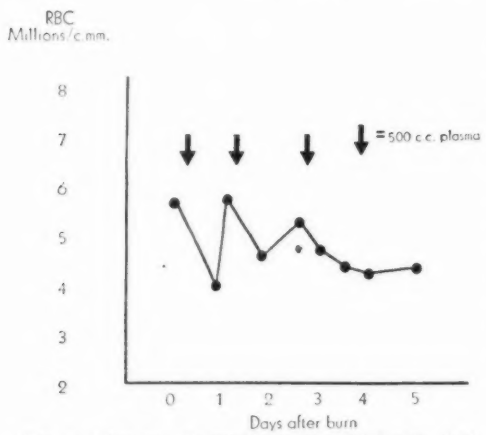

Fig. 5. Case 33. Twenty-four year old female, flash burn involving 30 per cent of the body surface, requiring some skin grafting. Complete healing by the 44th day. Note the persistent hemoconcentration permanently corrected only after three plasma transfusions.

labored and it was assumed that severe pulmonary damage had occurred, inasmuch as the child had been trapped in a burning house and undoubtedly inhaled a good deal of noxious gases as well as actual flame.

The remaining 2 cases (Cases 50 and 51) more than the others may be said to represent preventable failures in so far as the use of more plasma may have made a difference. However, pulmonary damage may have been partly responsible for the fatal outcome inasmuch as both patients were burned in the same accident, an explosion which occurred in a closed room and undoubtedly made inhalation of heated gases inevitable. Autopsy was done in only 1 case, but showed inflammation and deposition of soot down as far as the bronchi, as well as other evidence of pulmonary injury. Symptoms of circulatory impairment were present on admission in 1 case, but responded dramatically to a plasma transfusion, only to recur during the night. It was assumed to have been present for many hours before the second transfusion was started, because the patient died before very much plasma could be administered. In the other case (Case 50) surgical shock appeared only several hours after admission, and the patient died before transfusion could be started.

The frequency of vomiting, evidence of shock and toxemia in the fatal cases, should emphasize the importance of these clinical findings. These manifestations in many cases proved a more significant indication of the seriousness of the burns than the extent of skin involved. Moreover, all of the fatal cases were flame or flash burns. These factors are emphasized because they suggest that special study and treatment be accorded such patients from the beginning in order to save more of them if possible, particularly those involving a relatively moderate extent of the body surface.

COMMENT

On the basis of the present and previous studies, it has become increasingly evident that the systemic manifestations in severe burns including hemoconcentration, sensorial changes, circulatory impairment, and even fever and renal damage vary tremendously from case to case. Moreover, it seems clear that any attempt to correlate these differences with the degree of tissue damage must be based on more information than that given in the usual designations of extent of body involved and the terms first, second, and third degree burns. It would seem worth while evaluating carefully in each case the actual nature of the burn stimulus as well as its probable duration. The type of skin damage produced by various thermal injuries has received little study, particularly in fatal cases in which complete microscopic study of the skin is possible. Such information is needed if we are to explain the great variations in the systemic effects of severe burns and to save more of the fatal cases with as simple and effective therapy as possible.

Variations in the actual thermal injury may be classified into at least three groups: (1) The degree of *temperature* of the thermal agent may vary from 70 degrees C. in a hot water burn to several thousand degrees in a flash burn. (2) The *duration* of the increased temperature varies from a fraction of a second in some flash burns to many seconds in the case of hot water or flame; it undoubtedly lasted many minutes in the case of the victims trapped in the Cocoanut Grove or Hartford

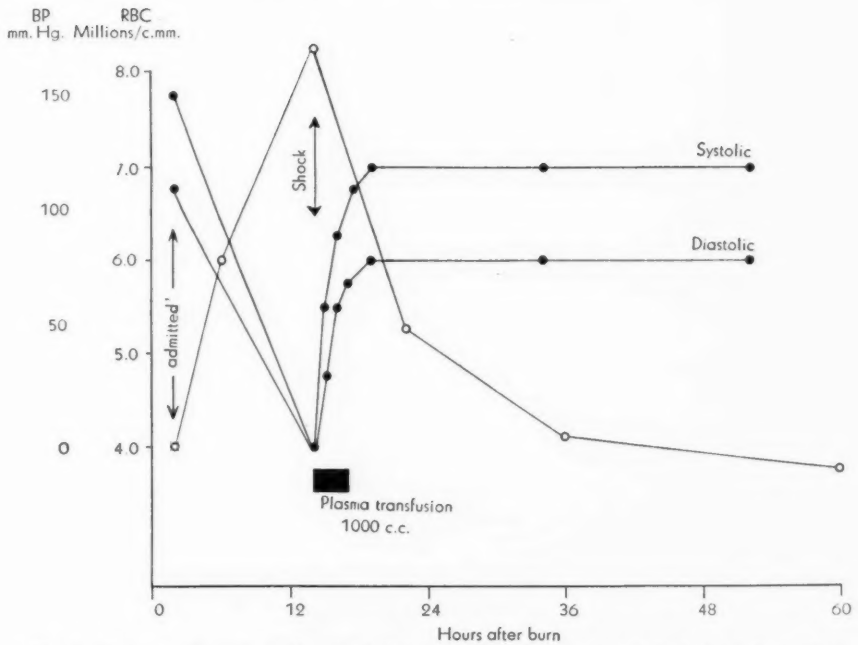

Fig. 6. Case 40. Seven year old male, flash burn involving 30 per cent of the body surface. Note that on admission there was absence of hemoconcentration and definitely elevated blood pressure. The general condition was good. After 12 hours typical surgical shock developed with cold extremities, rapid pulse and unmeasurable blood pressure. This was accompanied by hemoconcentration. A rapid plasma transfusion of 1000 c.c. (30 c.c. per kilogram) effected a dramatic clinical improvement, and as shown in the chart, a prompt restoration of blood pressure and correction of hemoconcentration. Skin graft was eventually necessary. ●—●, Blood pressure; ○—○, red blood cells.

circus fires. (3) The *part of the body* involved also contributes to variations as, for example, the relative resistance of the face as compared to the hands, due undoubtedly to the differences in their blood supply and tissue tension.

Classification according to the thermal agent itself, e.g., flame and hot water burns, may explain many of the differences. For example, in the present series none of the 21 hot water burns proved fatal and only two required skin graft, yet a third of them involved 30 per cent or more of the body surface. Moreover, more of them exhibited hemoconcentration than did the flame and flash burns. Nevertheless, all of the fatal cases were flame and flash burns and all but one showed hemoconcentration. In contrast to the relative infrequency of skin graft even with extensive hot water burns, two thirds of the surviving flame burns required skin graft even though many involved small areas of the body. In spite of the frequency of hemoconcentration in the hot water burns the incidence of fever was less pronounced, i.e., half of them showed none during their entire stay. By contrast, only a third of the flame and flash burns were afebrile during their entire stay. It should be emphasized that fever was only occasionally associated with other manifestations of infection; in most cases it was probably due to absorption from devitalized tissue or extravasated serum.

The satisfactory results which can be obtained with the simplest of dressing were well demonstrated in the present series of cases. Despite the hundreds of different ointments recommended for application next to the burned skin, it would seem that none is really required. Much more important is the meticulous avoidance of droplet and other

contamination during the dressing. The fundamental fact that much of the infection in burns can be traced to bacteria which reach the burn *after the patient enters the hospital* was established by Cruikshank over 10 years ago. Its truth and practical application in dressing burns seem amply shown in our findings.

The rather limited use of intravenous injections in the present plan of therapy demands special scrutiny. While such treatment is always complicated and should be minimized as far as possible in formulating a simplified plan of treatment, it should never be withheld when indicated. The most absolute indication is, of course, the possibility in combating, in the severe cases, any tendency toward a fatal outcome. How can one tell? The present observations have certainly indicated that many extensive burns will undoubtedly heal promptly and satisfactorily without such intravenous injections. On the other hand, there is always the possibility that fatalities may result from such a policy. From our observations as reported herein it is evident that constant observation is necessary during the first 24 to 48 hours in any patient with a flame burn exhibiting such systemic manifestations as vomiting, severe sensorial depression, actual or incipient circulatory impairment. These manifestations are of grave import even when the burn involves but 20 or 30 per cent of the body surface. Individualization must therefore be an important feature of any plan of treatment. Close scrutiny of such cases will undoubtedly minimize the mortality. It is in this group of cases that special methods of treatment may be indicated. For example, the injection of sodium lactate or bicarbonate may prove to be of decisive importance (5,13,14). In subsequent patients with such severe manifestations we expect to study changes in the carbon dioxide combining power and the effect of introducing bicarbonate or lactate solutions. The preference for whole blood over plasma (1,9,14) even though hemoconcentration is present may also prove decisive in this group of patients. It would seem obvious, however, that no method of therapy can be evaluated when used in extensive burns which would have done just as well without them.

With special emphasis on these potentially fatal cases which we believe can be detected shortly after admission, routine care of severe burns need not involve complicated methods of treatment. This is an important practical consideration. If the routine case can be handled in a relatively simple manner, attention and effort can be more effectively concentrated on the more serious ones in order to answer some of the unsolved questions, particularly regarding methods of therapy designed to further reduce mortality.

SUMMARY

A series of 55 severe burns (covering 10 per cent or more of the body surface) were treated by the application of a simple gauze dressing, without preliminary débridement, but with meticulous aseptic care, and left in place as long as possible, often 2 weeks. Plasma transfusions were not used as routine, but only when indicated by circulatory impairment; as part of the early treatment it was employed in 10 of the 43 healed cases. Morphine was not used as routine, but only for the relief of pain in 9 of the 55 cases. A high fluid and food intake was an essential feature of therapy and was possible from the beginning by the oral route in all but a few of the severe cases. The clinical findings in the 43 healed cases were satisfactory and showed relative absence of infection, rapid healing, and required a relatively small amount of skin grafting.

Analysis of the systemic manifestations shows tremendous variation and suggests that the extent or depth of the skin involved is not always an accurate indication of its seriousness. Of great significance was the kind of burn, which should be described as flame or scald, and, when possible, in terms of the duration and temperature of the burn stimulus. Analysis of the fatal cases suggests that they may be detected early by the presence of vomiting, shock, toxemia. In the face of such manifestations even in nonextensive burns special therapy is indicated to reduce mortality further in these cases.

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TECHNIQUE FOR THE REPAIR OF POSTOPERATIVE ABDOMINAL HERNIAS

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IT is not my intention to discuss the prophylaxis of postoperative abdominal hernia. The purpose of this communication is to discuss the surgical technique for the closure of the ring or opening when a hernia has unfortunately developed.

ANATOMICAL CONSIDERATIONS

For the purpose of this discussion the abdominal wall may be considered as divided into two parts, an upper and a lower, the level of the umbilicus being roughly the dividing line.

Thus a cross section of the abdominal wall at the various levels may be comparable to a bow with a taut string in the upper and to a bow with a loose string in the lower (Fig. 1A and B).

In the upper abdomen the wall is firm and tense and the strong lateral pull of the abdominal muscles and their costal attachments frequently make closure of vertical incisions difficult. Every surgeon has experienced the annoyance of the sutures cutting the tissues when closing the posterior sheath of the rectus muscle. When a hernia develops in this region, paradoxical as it may seem, the long axis of the ring is pulled transversely though the primary incision was vertical (Fig. 2A).

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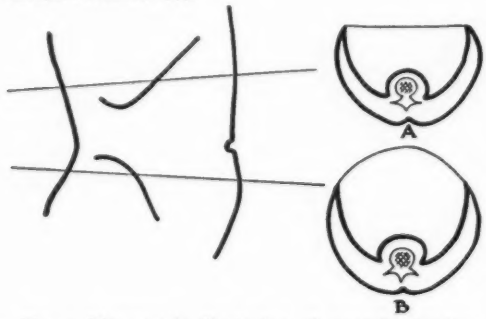

Fig. 1 Diagramatic illustration of muscular tension. A, Upper wall. B, Lower abdominal wall.

In the lower abdomen the wall is more lax, tends to bulge forward and downward, and in some cases becomes attenuated and even pendulous. Here the hernial opening is usually longer in the vertical diameter (Fig. 2B).

The linea alba is normally weaker in the lower abdomen. When a pendulous abdomen develops, usually associated with an umbilical hernia, the linea alba gives way to a point about 2 inches above the symphysis pubis. Professor I.M. Thompson, head of the department of anatomy in the University of Manitoba, stated during a discussion of this fact that: "The structures that reinforce the linea alba at this location are the pyramidalis muscles and their fibrous attachments to the linea alba and the reflected parts of the inguinal ligaments. These fibrous attachments undoubtedly prevent any further downward diastasis of the rectus muscles."

Furthermore, the diastasis of the rectus muscles stops abruptly above, at, or near the level of the umbilicus and this is where the lowest pair of the transverse intersections (linea transversae) of the rectus muscles is attached to the linea alba on each side. Thus with the distention limited above and below and laterally by the rectus muscles, the internal ring of a pendulous abdomen is well defined. These anatomical limits may be disrupted when a pendulous abdomen results from an incisional hernia.

TECHNIQUE OF REPAIR

Taking into consideration the anatomy of the abdominal wall and the structural changes that occur, four methods seem applicable to the various types of hernias that follow abdominal operations.

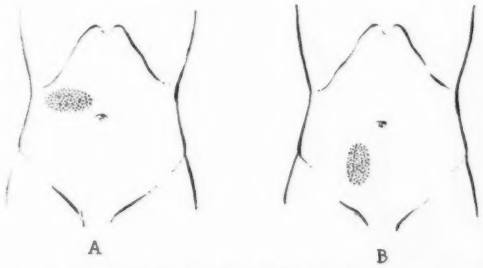

Fig. 2. Illustrating the shape of hernial ring resulting from muscular pull in A, the upper abdomen; B, the lower abdomen.

1. In the upper abdomen, the ring being longer in the transverse diameter, repair would logically be done transversely. This is applying the principle of the Mayo umbilical hernia operation to a postoperative hernia in this region. Arthur E. Benjamin has also applied this principle successfully in his work in suitable cases. I have frequently split the upper (usually the thicker) margin of the ring into two flaps and sewed the lower edge between them, thereby making stronger union (Fig. 3). As the posterior flap of the divided upper margin is necessarily sewed to the posterior surface of the lower there is less likelihood of omentum worming in between stitches as pointed out by Elliott Michelson and William Raffel.

2. In the lower abdomen the wall being more lax the problem of closure therefore is quite different. Here the hernial ring is a vertical oval and closure should be done vertically. The lateral margin of the ring, the thicker of the two, is split and the medial margin is stitched between these two lateral flaps, three layers thereby closing the opening (Fig. 4).

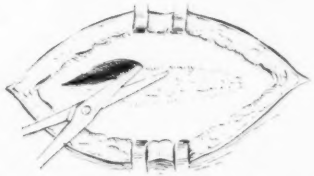

Fig. 3. In the upper abdomen repair is done transversely by overlapping the edges or splitting the upper and sewing the lower between the flaps formed.

3. At times the hernial opening is very large, as if the whole incision had separated and neither transverse nor vertical sewing seem to suit the case, the tension in the center being too great. In this event I have found the following expedient useful. Sewing is begun at the lowest point of the ring or opening and is continued upward to a point where tension is not too great and the sutured part can meet with the upper segment of the ring (Fig. 5A). The remaining part of the opening is then closed transversely resulting in a "T" shaped suture line (Fig. 5B).

In rare instances this procedure may be reversed, sewing beginning at the uppermost point of the opening. Only once in a very high hernia, following a paramedian incision which had completely disrupted, I found this latter method to be the only one that seemed suitable in this particular case (Fig. 6B). Where

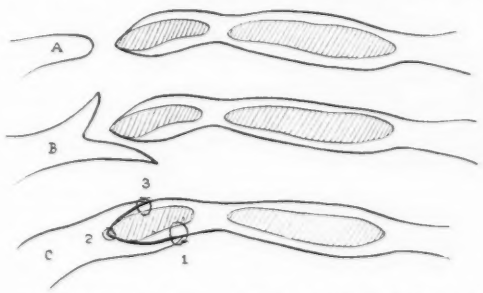

Fig. 4. Lower abdomen. A, Hernial defect. B, Lateral margin is split. c, Medial margin is sewed into the lateral cleft. The suture lines are not directly superimposed and they have a buffer layer between them.

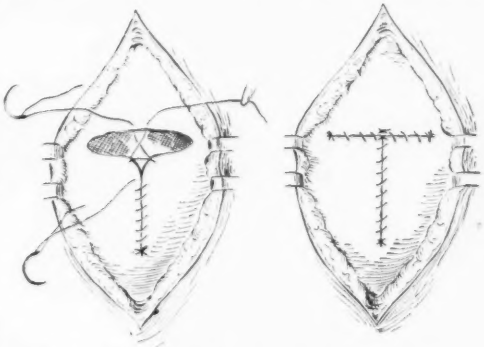

Fig. 5. A large circular ring may be closed by a combination of A. left, vertical; B, transverse sewing.

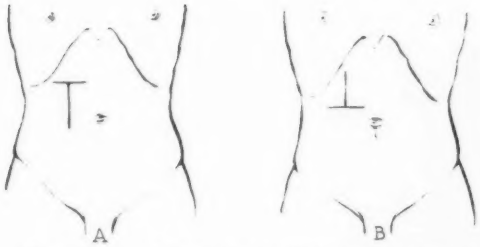

Fig. 6. Closure of a large ring partly from below upward A, or from above downward; B, depending on the muscular pull and ending the closure transversely. Only in an exceptional case would the second procedure be applicable.

overlapping cannot be done, figure-of-eight reinforcing stitches are used. These stitches are tied over a Wetherbee plate as this plate assures no give and there is no drag on the skin. If the stitches are tied without tension cutting or necrosis of the fascial edges should not occur (Fig. 7). These stitches remain in 14 or more days.

I would like to point out here that dissecting out the anatomical layers which have become fused together from the primary operation, as some advocate, is inadvisable. To do so prolongs the operation, unnecessarily traumatizes tissue with poor circulation and furthermore suture material is necessarily superimposed one row upon another which does not favor good healing.

4. In long standing cases in the lower abdomen the wall may be, as already mentioned, attenuated or even pendulous, especially if

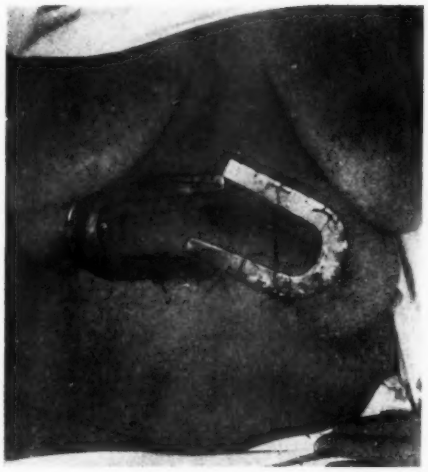

Fig. 7. Reinforcing figure-of-eight silkworm gut stitches tied to Wetherbee plates. The ends of two parallel stitches are tied together at one side of the plate and the opposite ends on the other side. The stitches do not pass over the skin wound or the plates. It is best to use double strand stitches.

the rectus muscles have become atrophied from the severing of nerves. For this type the three flap operation for the extreme pendulous abdomen that I described before this Association in 1917, seems best suited (3). The operation was originally designed for cases in which an umbilical hernia had piloted the protrusion so that the umbilicus (and its hernia) was no longer in its normal position in front of the abdomen but was 15 or 16 inches farther down

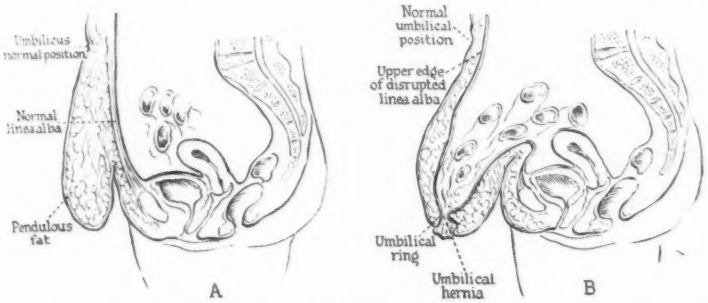

Fig. 8. Pendulous abdomen. A, Simple pendulous fat (panniculus adiposus). Note the abdominal wall is intact. B, The linea alba has gradually stretched into a funnel-like process. An umbilical hernia is at the apex, having descended far from its normal position. The funnel-like tubular process is filled with small intestine and omentum. Note: The defect does not reach the symphysis below and only to the normal umbilical level above. This is not a postoperative condition.

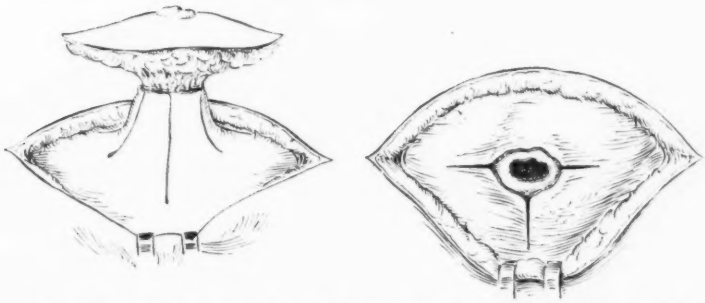

Fig. 9. First stage of a pendulous abdomen operation associated with an umbilical hernia. A, left, Removal of excess fat showing the cone of thinned out abdominal wall with the umbilical hernia at its apex. The flaps are outlined. B, Umbilical hernia removed and the three flaps formed. Note: The flaps are shown retracted. Diastasis of rectus muscles seldom occurs above the normal umbilical level, except in incisional hernias where the incision and disruption has gone above this level.

in front of the thighs (Fig. 8). With the passing of time I have not been able to improve on this method. Briefly the technique is as follows:

When the umbilical hernial ring has been cleared along with pendulous fat (lipectomy and herniotomy), the thin surrounding wall which can be lifted up in the form of a cone is dealt with (Fig. 9A).

I would like particularly to stress the point that in these pendulous abdomens whether associated with an umbilical hernia or an incisional hernia, there is always a second ring (in addition to the primary umbilical or incisional hernial ring) formed by the abdominal wall which did not take part in the protrusion, with a firm strong margin either circular or ovoid which might be called the internal ring of the hernia. Thus nature has provided an excellent framework to which flaps can be securely sewed. This is readily felt by inserting the fingers or hand into the sleevelike protrusion.

The lower margin of the primary ring is incised downward to a point about 2 inches above the symphysis pubes, or where the wall becomes firm and strong (see "Anatomical Considerations"—ante). Two lateral incisions are likewise made on each side outward to the rectus muscles, thus forming three flaps (Fig. 9B).

The lower two flaps are overlapped and stitched together making a double layer (Fig. 10A). The upper flap is now raised and the double lower flap is sewed to the firm tissue at its base (Fig. 10B).

The upper flap is now drawn down over the lower doubled flap and stitched in place, completely closing the defect with three layers (Fig. 10C).

J. C. Masson of Rochester has found this method applicable in incisional hernias where

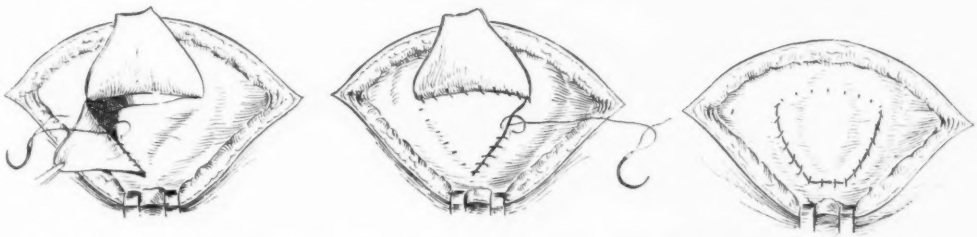

Fig. 10. A, left, Border of one flap is sewed to the posterior border of the opposite rectus sheath. B, center, Lower (now double) flap is sewed to the firm edge under the upper flap. This edge as well as the rest of the internal ring is always well defined and very firm. C, right, Upper flap is now stitched over the lower flaps and the repair is complete.

there is marked diastasis of the rectus muscles. This would necessarily apply to a hernia in the midline at or below the level of the umbilicus, but also it is applicable in hernias lateral to the midline where the ring is large and the wall thinned out and bulging.

The preoperative and postoperative treatment needs stressing. Preoperative preparation is directed toward (1) reducing weight, (2) reducing distention, (3) cleaning and sterilizing the operative field, and (4) treatment of avitaminosis and any secondary anemia. This preparation may in some cases take as long as 3 months.

The postoperative treatment consists of (1) flexion of abdominal muscles with pillows (not a straight back rest), (2) nasal gastric suction in all cases until the stage of distention is past and the early use of rectal tube and the judicious administration of prostigmine and pitressin, and (3) surgical fluids *per os* for 4 days or longer if necessary.

RÉSUMÉ

1. The type of operation for the repair of an incisional hernia varies for each case and has little to do with the type of the primary incision.

2. The shape of the hernial ring will determine whether closure shall be vertical or transverse.

3. Dissecting out the individual primary layers is unnecessary and complicates the operation. Overlapping is preferable or splitting the thicker edge with overlapping and underlapping is to be preferred. The simpler the procedure the better.

4. A large hernial ring may be closed by combined vertical and transverse suturing as described.

5. In a huge hernia with attenuated and pendulous abdominal wall the "Three Flap Operation" will, I think, be found most suitable.

6. Meticulous preoperative and postoperative treatment and care are a *sine qua non*.

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ONE STAGE TUBED ABDOMINAL FLAPS

Single Pedicle Tubes

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ROBERT L. PAYNE, Jr., M.D., Major, M.C., A.U.S., Norfolk, Virginia

WAR wounds of the upper extremity involving extensive loss of soft tissue frequently require pedicle replacement of skin and subcutaneous tissue. The need for early coverage and the desirability of expediting the treatment of a large number of cases has emphasized the usefulness of the direct abdominal flap for this purpose. Single pedicle abdominal flaps may be applied to any defect of the hand or forearm as a one stage procedure. The open pedicle flap as frequently used has the disadvantage of a contaminated wound requiring secondary closure. Any open wound of the hand impairs the return of function. Therefore, it is desirable to convert the donor area and base of the pedicle into a closed wound. This also diminishes the dressing problem and the possibility of infection at the time of division. The donor site may be sutured primarily or covered by a split thickness skin graft. In either case the base of the pedicle may be grafted. Whenever possible, the authors have closed the donor site as a linear incision, a simpler and more direct method which minimizes the subsequent deformity. The desirability of closing the base of the pedicle as well as the donor site has led to an endeavor to combine the advantages of the closed tube with the advantages of the direct abdominal flap. This has been successfully done in 31 cases by constructing one stage tubes, 25 of which covered defects of the hand and wrist.

It is possible to raise large, long, oblique, or vertical flaps from the lower abdomen and still close the donor area because of the greater mobility of the tissues in this region. When based inferiorly an excellent blood supply is assured by including the superficial epigastric and superficial circumflex iliac veins and accompanying arteries. These characteristics of size and viability permit easy tube formation. When combined with the adaptability of the hand to various positions, the usefulness of these tubes for covering irregular defects of the hand becomes apparent.

Single pedicle tubes, based inferiorly, including the superficial epigastric veins have been constructed, varying in length from 5 to 18 centimeters and varying in width from 3 to 7 centimeters. Exact limitations of size have not been determined. The dimensions depend on the size of the defect to be covered and on the obesity and mobility of the abdominal tissues. The superficial veins can usually be seen through the skin and are used

Fig. 1. Infra-red photograph showing superficial epigastric veins (left), and donor area where tube was recently detached (right).

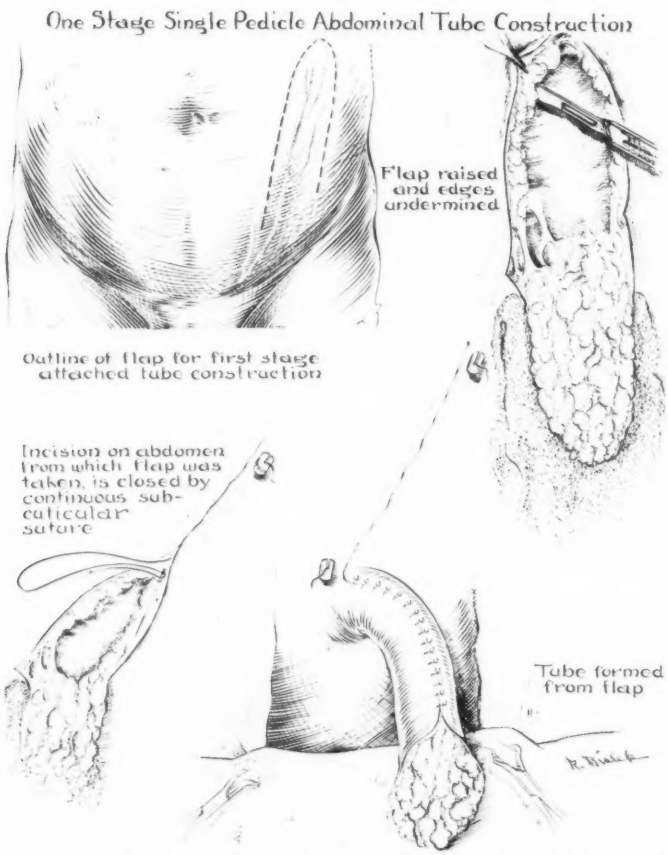
One Stage Single Pedicle Abdominal Tube Construction
Flap raised and edges undermined
Outline of flap for first stage attached tube construction
Incision on abdomen from which flap was taken, is closed by continuous subcuticular suture
Tube formed from flap

Fig. 2. Diagram of tube construction showing inclusion of superficial epigastric veins, undermining of margins for closure of donor area, and closure by continuous subcuticular stainless steel wire suture.

as a guide in outlining the flap. If the veins are not visible their approximate location can be estimated.

The flap is outlined on the abdomen as two parallel incisions tapering to a blunt point superiorly. It is raised either just superficial to Scarpa's fascia or may include the fascia. The margins of the donor defect are undermined for closure in the fatty areolar layer beneath the fascia. Additional relaxation may be obtained by incision of the fascia parallel to the skin margins. The attachment of the flap is extensively undermined to allow mobility in closing the angles when the base of the tube is formed. The donor defect is closed with continuous subcuticular stainless steel wire. This procedure has proved to be a rapid, simple, and satisfactory method of closure and incorporates the basic principle of using a buried subcuticular suture for closing skin under tension. This continuous suture includes one or both angles of the incisions at the base of the flap and begins formation of the tube. By staggering the inferior ends of the incisions the base of the tube may be rotated through an arc of 180 degrees. If the medial incision is shorter the open surface of the tube will rotate laterally. If the lateral incision is shorter the tube will be rotated medially. The angle of rotation depends on

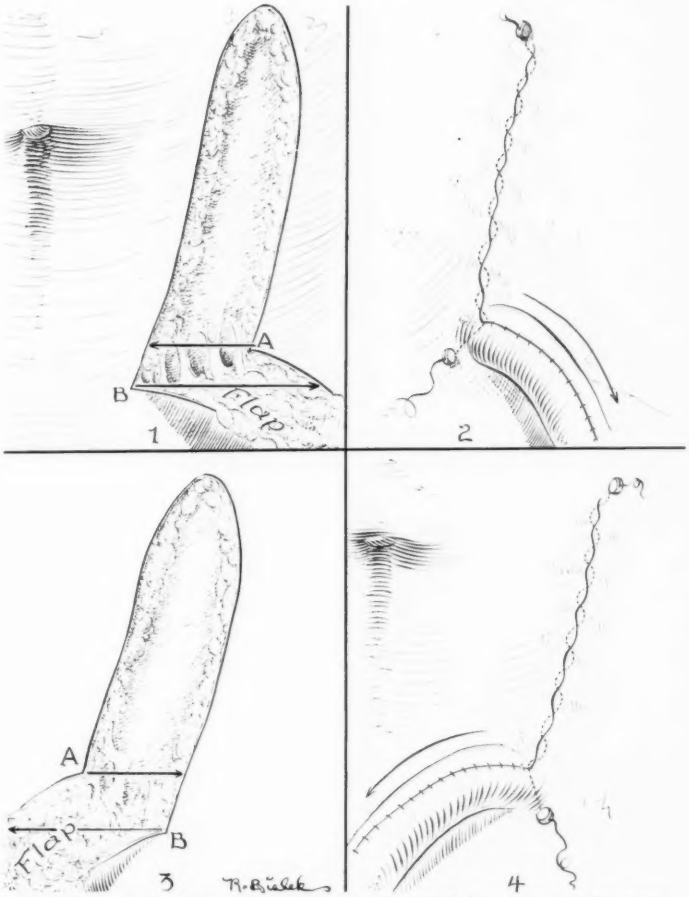

Fig. 3. Diagram showing rotation of tube to right or left, by staggering of incisions.

the amount of staggering of the original incisions and on the width of the flap. Thus the open surface of the tube may face medially, superiorly, or laterally, without twisting the tube itself. Combined with the fact that it may be folded on itself, application to the hand is permitted in a wide variety of positions.

The flap is formed into a tube by simple interrupted sutures and is applied to the recipient defect by subcuticular sutures of fine, white, twisted nylon and interrupted skin sutures of fine stainless steel wire or fine silk. The wire seems to give less reaction than does silk in the presence of moisture.

If the original defect is completely covered at the time of application, the tube may be handled much the same as is an abdominal flap and divided in about 3 weeks. However, if the tube itself is used to cover a portion of the defect, a longer period of time is allowed and some type of delay is advisable, depending upon the length of the tube. When the tube has a small application to the recipient area, as when it is attached to the wrist to be carried to some other portion of the body, or used in total thumb reconstruction, the time of application must be longer than when a double pedicle tube that has been constructed previously is applied. This difference in time we attribute

Fig. 4.

Fig. 5.

Fig. 6.

Fig. 7.

Fig. 8.

Fig. 10.

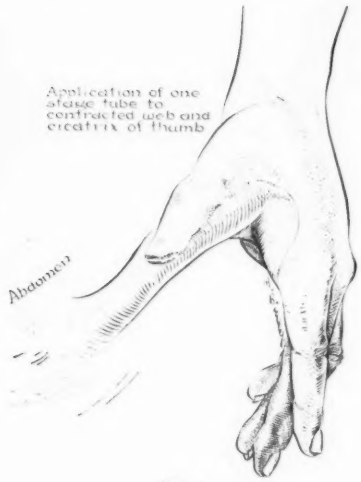

Fig. 9.

Fig. 11.

Fig. 12.

Fig. 13.

Fig. 4. Case 1. Preoperative view of severe scar and contracture which was present between the first and second metacarpals.

Fig. 5. Case 1. Postoperative view following excision of scar, release of contracture, and application of one stage single pedicle tube.

Fig. 6. Case 1. Dorsal view of applied flap.

Fig. 7. Case 1. Palmar view of applied tube.

Fig. 14.

Fig. 15.

Fig. 16.

Fig. 17.

Fig. 18.

Fig. 19.

Fig. 8. Case 2. Web contracture associated with scar on palmar surface of amputated thumb.

Fig. 9. Case 2. Diagram of application of tube.

Fig. 10. Case 2. Postoperative view showing correction of contracture and application to thumb.

Fig. 11. Case 3. Tube to correct contracture after amputation of middle finger with palmar and dorsal scar.

Fig. 12. Case 4. Tube applied to wrist.

Fig. 13. Case 5. Tube applied to palmar surface of finger to correct severe flexion contracture. When the tube was divided the end was applied to correct the cicatrix of the amputated thumb.

Fig. 14. Case 6. Tube applied for reconstruction of thumb.

Fig. 15. Case 7. Tube applied to palm.

Fig. 16. Case 8. Another case in which the tube applied to palm.

Fig. 17. Case 9. Tube applied to dorsum of hand.

Fig. 18. Case 10. A type of flap in which tube formation may be a disadvantage. Because of the width of the attached margin, tube formation was not desirable and the base was covered by a split thickness skin graft.

Fig. 19. Case 11. Tube attached to wrist to be carried to another part of the body.

to the primary healing reaction and unestablished blood supply in the one stage tube. Compared with the double pedicle tube the time of application is longer but the total time is shortened.

Delaying tubes, which not infrequently have a border line blood supply, is best done as a closed procedure in order to prevent superimposed infection which may further impair the blood supply, in this manner forming a vicious cycle which may result in partial loss of the tube.

SUMMARY

One stage single pedicle abdominal tubes have been found a practicable and an advantageous method of applying skin and subcutaneous tissue to defects of the hand. They combine the speed of the abdominal flap with the cleanliness of the tube. When attached to the wrist they may be carried to other parts of the body.

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THE EFFECTS OF CERTAIN DRUGS ON THE SPHINCTER OF ODDI

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PRESSURE changes in the biliary system secondary to variations in tone and states of contraction of the musculature surrounding the lower end of the common bile duct have been shown to occur in many animals and in man. Former investigations have demonstrated changes in intraductal pressure by measuring these variations, which have been relayed up through a cannula in the common duct and into a manometer system. Since direct observations of the sphincteric contractions of this muscle system had not been recorded, it was believed that their measurement might throw additional light on the reactability of the sphincter of Oddi.

LITERATURE

Oddi was the first to measure the intraductal pressure. He measured the tonus of the sphincter by observing the height of a column of water supported in a tube tied in the common duct. Doyon reversed this procedure by measuring the amount of fluid passing through the ampulla from a specially constructed manometer connected with the common duct.

The effects of external stimuli and diet on intrabiliary pressure have been studied by several observers. Archibald noted that touching the duodenal mucosa with hydrochloric acid or pinching it raised the tone of the sphincter so that pressures as high as 800 millimeters of water were recorded. McWhorter found that magnesium sulfate in the duodenum lowered the pressure produced by the tone of the sphincter from 50 to 100 millimeters of water. Jacobson and Gydesen observed that in addition to magnesium sulfate, acids and peptone lowered the sphincter tone. Cole found that isolated gastric contents influence the pressure variably. Bruno, Klodnizki, Boldyreff, and Rost observed intermittent gushes of bile from the ampulla of unanesthetized dogs which they interpreted to be the result of the relaxation of the sphincter. This observation has been confirmed many times by others (Puestow; Klee and Kluepfel).

Lueth studied the effects of various drugs on the intraductal resistance in the common duct and found that in general the effects on the intraductal resistance and duodenal tone were similar.

The pressure necessary to cause a flow of water from the common duct through the ampulla has been measured by many observers. In the dog it has varied from 60 (Winkelstein and Aschner) to 675 (Oddi) millimeters of water. In man most observers agree that the normal tonicity of the sphincter of Oddi varies between 90 and 150 millimeters of water (Butsch, McGowan, Walters; and Elman and McMaster, 9, 10). This resistance may be raised from 600 to 700 millimeters water under certain circumstances.

METHOD

External biliary fistulas were prepared in dogs according to Puestow's technique. The major ampulla was transplanted to the skin after intestinal continuity was restored. The first operation consisted in an anastomosis such as is carried out in a posterior Pólya resection of the stomach. When the dog had recovered, the closed duodenal loop was transplanted between the abdominal muscles and the skin. At the final operation, 7 to 10 days later, this loop was opened and cut across between the openings of the pancreatic and common bile ducts. The lower opening into the bowel was inverted and the skin closed over it. The edges of the upper opening were attached to the skin so that the ampulla was easily accessible. Over a period of months it was necessary to raise this portion as it tended to retract into the abdominal cavity.

From the Laboratory of Surgical Research, Western Reserve University, Cleveland, Ohio.

The dogs were fed on a standard diet with cereal and meat scraps. On this standard regimen they were kept alive for months. The two dogs on whom most of the readings were made stayed alive 215 and 250 days, respectively. The most dangerous complication exclusive of distemper was the opening of the lower loop, causing complete loss of pancreatic secretion, maceration of the skin, and a rapid downhill course. These dogs were thus kept alive with complete loss of bile except what they licked from the fistula. The mucosa around the fistula on manipulation bled easily, but only one dog showed any evidence of gross bleeding at autopsy.

After complete recovery the dog was strapped on his back on a table four or five times weekly for a half hour until he learned to lie quietly. All readings were made in the fasting stage. A small latex rubber bag with a hard tip and measuring 3 millimeters in diameter was inserted through the ampulla for a distance of about 2 centimeters. The proximal end was attached by glass and rubber tubing to the recording device. At first this method consisted of a rubber tambour and the conventional writing point on a smoked drum. The recorded excursions were so small that a more sensitive method which would increase the deviations was sought.

A photoelectric method by means of the electrocardiographic apparatus was devised. A beam of light separate from the electrocardiograph was reflected from a projection cell onto the photographic paper. Any variations in pressure on the rubber bag were transmitted to the cell, thus causing deflections of the light beam. A closed air system was used throughout the experiments. Since the speed of the photographic paper past the exposure slit was slowed by adding resistance in the electric circuit controlling the motion, variations in this speed were noted, caused by

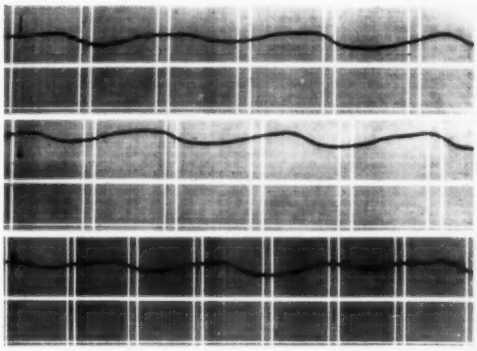

Fig. 1. First record, control reading; second record, following inhalations of amyl nitrite for 1 minute; third record, 5 minutes later.

changes in electrical resistance and friction of the rotating belt. To make the results comparable a timing device was added. A metronome was placed in front of the beam of light used in normal electrocardiography so that the moving pendulum would cut the beam. Because of the construction of the electrocardiograph the metronome could not be put exactly in front of the light beam, with the result that two vertical white lines close together caused by the metronome pendulum cut the light beam at the end of one and at the beginning of the next arc. Accordingly, the time interval between any comparable two lines is two seconds, the metronome being set at 60 oscillations per minute.

The curves shown on the photographic paper are the recorded contractions. The direction of the contractions is denoted by the arrow. The horizontal white line is the baseline used in electrocardiography and has no relation to the present records. No attempt for a standard baseline was made, since a "normal" or basal reading was taken at each period of observation for 10 to 15 minutes before any stimulus was used. Various stimuli

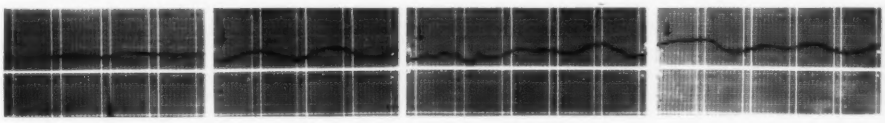

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Fig. 2. First record, control reading; second record, following 1/6 grain morphine sulfate subcutaneously; third record, during vomiting. Note change in second time interval; fourth record, 5 minutes later.

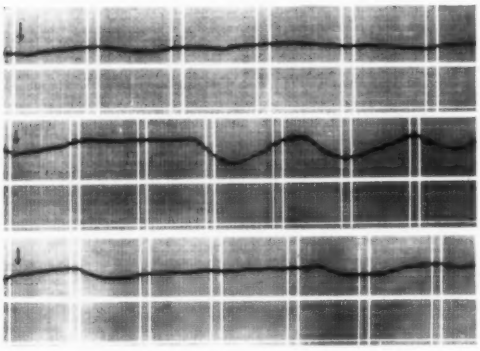

Fig. 3. First record, control reading; second record, following 1 cubic centimeter of surgical pituitrin subcutaneously; third record, 5 minutes later.

were used. Observations were limited to one a day so the effects of one stimulus would be gone when another was given. The stimulus was administered, an immediate reading taken, and a final reading again at 5 minutes. Repeat observations on the same animal under the same conditions were made as control procedures. The resulting curves were recorded, the paper was developed, dried, and representative portions were mounted on cardboard.

A standardization of the contractions showed that a difference of 1 millimeter on the recording paper equals 2 centimeters of water pressure.

OBSERVATIONS

Normal. A comparison of the control readings, i.e., those taken before any drug was given, shows that a slight rhythmic contraction occurs every 2 to 6 seconds. Each contraction was slow, taking from 1 to 2 seconds for the full variation in pressure to be recorded. These variations ranged from 2 to 7 millimeters, the average being 3 millimeters. During vomiting the pressure increased in one instance 12 millimeters or 24 centimeters of water pressure. This same change in pressure occurred in a much slower rhythm during excitement.

It is interesting that the "normal" or control readings vary from day to day in tone, rhythmicity, and number of contractions.

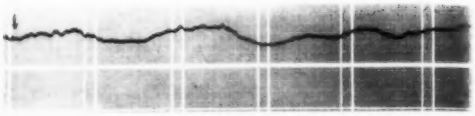

Fig. 4. Record taken during excitement.

Calcium. The dog was given 10 cubic centimeters of 5 per cent calcium chloride intravenously after a control reading was taken. There occurred a slight increase in tone, with no change in the amplitude of the contractions, and no change in the number of contractions.

Houssay and Rubio have stated that calcium chloride caused contraction of the gall bladder. Erbsen and Damm report that the calcium ion causes relaxation of the isolated guinea pig's gall bladder. The action of calcium on isolated smooth muscle is variable, although in the main it tends to cause relaxation. The results obtained here indicate that calcium increases the tone of the smooth muscle surrounding the lower end of the common bile duct.

Novocain. The topical application of 5 per cent novocain to the exposed ampulla had no observable effect on the tone, amplitude, or number of contractions.

Adrenalin. A subcutaneous injection of 1 cubic centimeter of 1:1000 solution of adrenalin caused an increase as high as 4 millimeters in the amplitude of the contractions, but no change in the tone or number of contractions. A dose of 2 cubic centimeters caused a still greater increase in the amplitude of the contractions to as high as 8 millimeters, an increase in the respiratory rate, and some restlessness in the animal.

Lueth reported a decreased duodenal pressure and intramural resistance after giving 1 to 5 cubic centimeters of 1:1000 epinephrine. With smaller doses the effects were inconsistent. In small doses the action on the gall bladder has been reported as variable. Larger doses caused contraction of the gall bladder. The amounts given in the present experiments were large, the effect being in proportion to the quantity of drug given.

Amyl nitrite. The dogs were caused to inhale the fumes of amyl nitrite for 1 minute.

The only effect on the state of the muscle tested was a slight decrease in tone (Fig. 1).

The effect of nitrites on smooth muscle is definitely one of relaxation. It is interesting, therefore, that while the tone of the muscle was reduced, the amplitude and number of contractions were unaffected. In view of the clinical relief which was obtained in patients with biliary dyskinesia, this finding was to be expected.

Hydrochloric acid. Tenth normal hydrochloric acid was applied topically to the ampulla as it lay exposed. The slight decrease in tone which occurred disappeared in one set of experiments and almost disappeared in the other during the observation period of 5 minutes. In one experiment the amplitude of the contractions increased slightly. Puestow noted a fairly constant stimulating effect on duodenal contractions and occasionally small spurts of bile occurred when dilute hydrochloric acid was applied. Elman and McMaster (9) noted a marked decrease in intraductal pressure when acid was introduced into the duodenum, while alkali caused a similar increase.

Morphine sulfate. One-sixth grain morphine sulfate was administered to the animal hypodermically. The frequency of the contractions increased slightly. The amplitude of each contraction increased from 3 to 6 millimeters, while a negligent change in tone occurred (Fig. 2).

Since morphine causes vomiting in dogs, several records during this action were taken. The musculature would relax until the actual vomiting occurred, when a sudden spasm would occur. Relaxation followed immediately. The time required for this was from 3/5 to 1 second. The pressure increased as much as 12 millimeters or 24 centimeters of water. No protracted spasm as reported by Butsch, McGowan, and Walters was noted. How much of this contraction was due to duodenal muscles could not be determined.

Pituitrin. One cubic centimeter of surgical pituitrin was injected hypodermically (Fig. 3). A definite and marked decrease in muscular tone occurred. The amplitude of the contractions increased up to 15 millimeters, while the number of contractions remained about the same. The record also shows many smaller contractions occurring, about 6 to 8 per minute. These could not have been the effect of respiration, because the respiratory rate at the time was between 30 to 40 per minute. In comparison to the normal tonus rhythm they could be described as fibrillary contractions. They occur in some of the other records in a less marked variation and may be artifacts.

The decrease in tone agrees with the findings of Lueth, who found an initial decrease in intramural resistance followed by a slow return to the control level.

COMMENT

The intraductal pressure at the level of the sphincter is an aggregate of four components. These consist of the secretory pressure from the liver, the pressure dependent on the state of the musculature of the gall-bladder wall, the compression exerted by the muscle bundles, both circular and longitudinal, surrounding the lower end of the common duct, and the back pressure exerted from the lumen of the duodenum when the ampulla is patent. In the experimental set-up which is here reported, all except the compression factor of the sphincter and duodenal muscles have been eliminated.

In the operative procedure used in these experiments the circular muscles of the duodenum have been cut so that any remaining constricting action is a purely local contraction. The recorded differences then are caused more probably by the action of the sphincter, and the large changes in pressures reported by other observers have been due to the combined action of the duodenal musculature and the sphincter.

The greatest variation from the control pressure was 7 millimeters and the greatest variation under any circumstances recorded was only 15 millimeters, or about 30 centimeters of water pressure, as computed by standardization procedures. After similar operative procedures and cholecystectomy, Puestow reported 20 to 30 centimeters of bile pressure in the common duct. Ivy states that in the unanesthetized fasting dog the intramural resistance may vary from 9 to 25 centi-

meters of water pressure, depending on the length of the fast. This variation coincides with the present finding. These low pressures may have been the result of several things.

There is little doubt that most, if not all, of the vagal fibers were divided during the operative procedures. The sympathetic nerves around the hepatic artery remained intact, however. Since the exposed duodenal segment seemed to recover a normal tone, the ultimate effect of division of the vagus seems questionable. Under barbital anesthesia, stimulation of the peripheral end of the cut right vagus nerve in the neck caused an increase of 4 centimeters of water pressure. Stimulation of the central end of the right vagus and both ends of the severed left vagus had no effect upon the contractions of the sphincter.

In addition to the nerve section, the insertion of the bag through the ampulla may have partially paralyzed or fatigued the sphincter, so that complete recovery had not occurred before the period of recorded observations. If this is true, then the pressure differences recorded are only a fraction of the real changes which occurred.

The length of observation was usually about 30 minutes. In certain experiments there is suggestive evidence that the effects of the drugs became apparent only after the 5 minute interval. However, readings continued beyond this period became complicated by restlessness on the part of the dog, so that the criticism of possible incomplete observation must be accepted (Fig. 4).

In acute experiments with the ampulla raised to the skin and using the same pressure recording apparatus, a normal tone of 0.5 centimeter of water was found. This was a maintained pressure in spite of the barbital anesthesia. This procedure was not done on the unanesthetized dogs, but it seems reasonable to assume that the normal tone in those animals would be higher. Lueth found the pressure necessary to open the sphincter to vary from 3 to 5 centimeters of water, which is probably normal tonic resistance.

SUMMARY

In dogs with a biliary fistula preparation which excluded all pressure factors except that which could be ascribed to the action of the sphincter of Oddi, studies of the changes in pressure in response to various drugs were carried out. The animals survived for many months (1 for 250 days) under normal conditions, so that observations could be carried out uncomplicated by anesthesia or trauma. In the fasting animal the bile flowed only rarely and the exposed segment of bowel was quiet. The sphincter normally underwent a more or less rhythmical slow contraction every 2 to 6 seconds, showing a pressure variation of 4 to 14 centimeters of water. The definite changes observed may be summarized as follows:

Calcium chloride administered intravenously caused a slight increase in tone.

Hypodermic adrenalin chloride, 1:1,000, increased the amplitude of the contractions.

Topical 5 per cent novocain showed no effect.

Inhalations of amyl nitrite resulted in a slight decrease in tone.

Tenth normal hydrochloric acid applied topically caused a slight transient decrease in tone.

Morphine sulfate given hypodermically increased the number and amplitude of the contractions. A marked increase in amplitude occurred during the act of vomiting. This is interpreted as a defense mechanism against reflux of duodenal contents into the biliary system.

Surgical pituitrin hypodermically caused a marked decrease in tone and a marked increase in the amplitude of the contractions.

These results in pressure changes are comparable, though in decreased values, to those reported obtained by methods of cannulating the common duct. Because of the method here reported, it is believed that these pressures more nearly represent the action of the sphincter of Oddi.

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LIPOSARCOMA OF THE FEMALE MAMMARY GLAND

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LIPOSARCOMA of the mammary gland is a rare tumor form in this organ, and the few cases which have been reported contain sparse and conflicting information about its degree of malignancy. Some of the cases have proved truly malignant producing metastases and causing death while others have pursued a much less noxious course. It is therefore by no means simple to decide how to treat liposarcoma of the breast when confronted with one. Because of this we propose to present the evidence of previously recorded cases, describe 1 new case, and discuss the question of treatment.

In Geschickter's monograph on diseases of the breast, mention is made without details of 3 women between the ages of 52 and 65 years, each of whom had rapidly growing breast tumors measuring 8 centimeters or more in diameter. All progressed rapidly to a fatal termination. He states that they can metastasize to the lymph nodes and that they are radiosensitive but not easily cured by this form of treatment. This exceedingly gloomy picture is duplicated by Lifvendahl's case. This was a 41 year old woman who noted small masses in both breasts. Three months later when she was examined there were 10 nodules in the left and 15 in the right breast. Bilateral mastectomy was done. The nodules were grayish-white and slimy. The largest measured 5 by 4 by 0 centimeters. Three months later she died with evidence of pulmonary metastases. It was an undifferentiated mucoid liposarcoma with many bizarre lipoblasts.

Adair and Herrmann cite briefly 4 cases from the Memorial Hospital, New York. Three of them were called myxoliposarcomas of low grade malignancy. One of these had numerous recurrences following simple mastectomy during a 10 year period and ultimately died of pulmonary metastases. Of the other 2 patients, one had local excision, the other radical mastectomy, and both remained free of disease respectively 4 and 4½ years after operation. The fourth patient was said to have had a "grade 4 liposarcoma" and lymphatic leucemia. She had no evidence of sarcoma 4 months after radical mastectomy.

Other cases have not pursued such a malignant course. Delage and Massabiau described a 52 year old woman who had a breast tumor weighing 1700 grams removed. It was called a myxolipoma. Six years later there was a recurrent nodule below the scar. Ten years after the first operation there were 5 nodules in the breast which the patient said felt like a bag full of oranges. A mastectomy was done and the tumors again called myxolipoma. Deaver and McFarland believe that this in fact is a case of liposarcoma and we agree with them. Neumann's patient was 60 years old. She had a slimy tumor in the inner hemisphere of the left breast. It was excised but recurred and 1 year later the new tumor was cut out. Recurrence followed and the third excision was done after 10 more months. It was a myxoid tumor with many cells containing fat droplets. In its last manifestation it was as large as an apple and reached but did not invade the corium. Neumann called it a myxoma lipomatodes but it seems proper to consider this also a liposarcoma of the myxoid type. Merkel's patient was 20 years old. She had a tumor at the margin of the gland which extended outside of it and in 6 months reached a size of 9 by 5.5 by 3.5 centimeters. It was encapsulated and had foam cells in it some of which were of giant size. Merkel called it a pseudolipoma but Borst saw the sections and considered it a true lipoblastic sarcoma. The final result is not recorded.

Fox's patient was 58 years old when a complete mastectomy was done because of an encapsulated tumor in the breast which had been present for 2 months. No metastases were found in the nodes and the patient was well 5 months later. It was called a liposarcoma but no other details are given. Cheatle and Cutler also record the presence of a small liposarcoma in the female breast which looked like embryonal fat. The photomicrograph shows many rounded foam cells, some with large bizarre nuclei. No other details were recorded.

There are 2 other cases in the literature which seem to be examples of liposarcoma developing in the stroma of cystosarcoma phyllodes or adenofibrosarcoma. In Binkert's case a man's head sized tumor, weighing 3 kilos, grew in the left breast of a 39 year old woman in less than a year. A radical operation was done and axillary metastases were found. In 5 months there was a recurrence in the scar and metastases in the opposite

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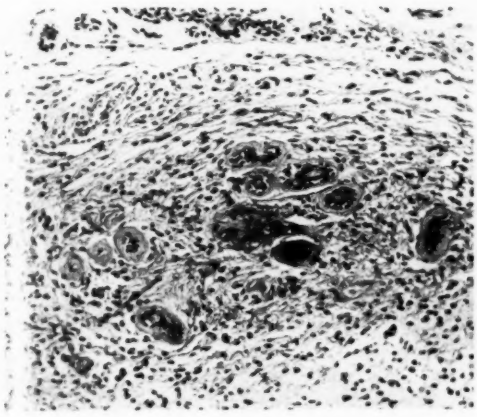

Fig. 1. Liposarcoma of the breast. Photomicrograph of the margin of the tumor showing infiltration around the eroded ducts and acini.

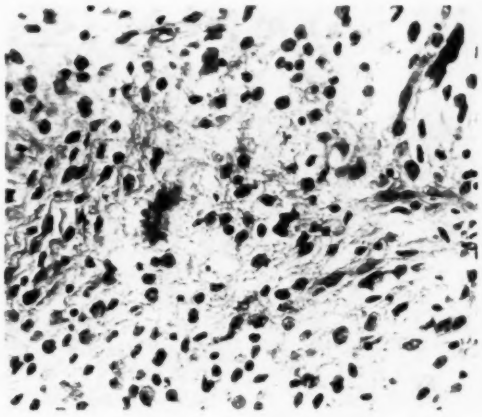

Fig. 2. Liposarcoma of the breast. Detail photomicrograph showing myxoid stroma and bizarre giant cells. Some of the cells are vacuolated.

axilla. A right radical mastectomy was done with removal of the nodule in the left-sided scar. The primary tumor was an adenofibrosarcoma (i.e. cystosarcoma phyllodes) with liposarcomatous stroma in places. The recurrence and metastases were liposarcomas with many bizarre giant cells but without epithelial elements. Binkert called the tumor a "fibrolipoadenoma intracanaliculare sarkomatodes xanthomatodes mammae."

A case somewhat resembling Binkert's has been reported by Hinterberger. A 25 year old girl noticed a cherry sized tumor in her right breast which in a year attained the size of an orange. It was excised and during the next 6 years, 5 more operations for the removal of recurrences were carried out. The earlier tumors were fibroadenomas with sarcoma-like stroma. In the fifth specimen there was lipoblastic tissue between the recurrent sarcoma phyllodes nodules. The tissue from the last operation was studied in detail by Hinterberger. Now there was still a cystosarcoma phyllodes nodule but other nodules were lipoblastic tissue which invaded the surrounding mammary tissue. There were never any metastases observed. The final result is not reported.

It is of passing interest to note that liposarcomas have been described in the mammary glands of cats, dogs and guinea-pigs (Corsy and Thomas, Schultz-Brauns; lit.)

CASE REPORT

A married Jewess, aged 43 years, noticed a lump in the left breast on December 5, 1945. She consulted one of us (M.B.) about it the next day. Menstruation had been regular and the past history uneventful. Examination showed a freely movable tumor 2.5 by 1.7 centimeters in the upper outer quadrant of the left breast. It was not attached to the skin and the axillary nodes did not feel enlarged. Roentgenograms of the chest and skeleton were negative. Sedimentation rate, Wassermann, and blood smears were normal.

Operation by one of us (M.B.) at the West Side Hospital on December 8, 1945, consisted of wide excision of the tumor and surrounding breast tissue. The wound healed well and there was no evidence or recurrence 2 months after operation. Gross examination of the specimen showed a mass of breast tissue 5 by 3 centimeters. Embedded in the center of this was a localized circumscribed tumor measuring 10 by 12 millimeters. It was soft, fleshy, and light brown in color.

Microscopic examination showed a tumor made up of a conglomeration of cells of varying size and shape, including round, spindle, and stellate forms and a number of bizarre multinucleated giant cells. These were set in a fibrous stroma some of the fibers of which were thick and others extremely delicate producing a myxoid appearance, although the mucicarmine stain showed no pink or red color. A scharlach R stain showed lipoid droplets in some of the cells. The tumor was circumscribed but at its periphery invaded the surrounding breast tissue to a limited extent (Figs. 1 and 2). The tumor was considered a partly differentiated myxoid liposarcoma.

It is apparent from a study of reported cases that liposarcoma of the breast is a tumor which sometimes behaves in a most malignant fashion, metastasizing both through the lymphatic and blood streams and resulting in death. In other cases the infiltrative nature of the tumor is shown by repeated local recurrences after incomplete excision occasionally with a long free interval between excision and reappearance but without any metastasis, and finally there are cases apparently cured by adequate simple excision of tumor and surrounding breast tissue.

A study of a considerable number of liposarcomas led one of us (A.P.S.) to the conclusion that they could be divided into two main classes, those which show a considerable degree of differentiation and which do not metastasize as long as the whole tumor maintains its good differentiation and a second larger class which is poorly differentiated, may show various types of metaplasia, and which metastasizes in about 40 per cent of cases. A reasonable form of treatment for the first group was considered generous excision, because even if this was followed by recurrence, metastasis need not be expected; whereas in the second group, a more radical procedure should be carried out because of the potential danger of metastasis.

The case here reported was unusual because it was small and was generously excised within a few days of its discovery. It was felt that it could be placed in the more differentiated group and that therefore metastasis was extremely unlikely and that if local recurrence should occur after the generous excision of tumor and surrounding breast tissue, it would still be possible to do a mastectomy at a later date without jeopardizing the woman's ultimate chance of cure. For these reasons no further operative procedures were carried out. Had the liposarcoma belonged to the poorly differentiated group of liposarcomas, a radical mastectomy would have been the proper treatment since such tumors may metastasize both through the blood and the lymphatic vessels.

SUMMARY

A new case of liposarcoma of the breast is recorded, previously reported cases are analyzed, and the treatment of this rare mammary tumor form is discussed.

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DEFORMATIONS OF THE SKULL IN HEAD INJURY STUDIED BY THE "STRESSCOAT" TECHNIQUE, QUANTITATIVE DETERMINATIONS

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DEFORMATION of the skull due to hammer blows has been studied by the "stresscoat" technique (3). If the tensile deformations are of sufficient magnitude they cause cracks in a strain-sensitive brittle coating previously applied to the surface of the skull. The resultant deformation patterns may then be studied in the immediate vicinity of, as well as remote from, the region of the blow. "Stresscoat" (1,2) is the trade name of a brittle lacquer method of strain determination in any material subjected to static and dynamic loads. The surface of the structure is coated with a lacquer. Cracks form in this coating when the material to be tested is subjected to tensile strains of 0.0005 to 0.0015 inch per inch. The sensitivity of the lacquer depends upon the temperature and humidity conditions at the time of the tests. Different lacquers are used for varying temperature and humidity conditions. Before a test, the sensitivity of a given lacquer is determined by a calibrating device.

Dogs and monkeys were used in order to establish a correlation between the strain patterns obtained in the skull of the living animal under nembutal anesthesia, the skull of the dead animal with intracranial contents undisturbed and in the dry skull of the same animal (3). It was found that the patterns obtained in the dry skull were less extensive than in the skull of the dead animal or in the animal under anesthesia, but the direction and general distribution of the strain paths were the same in all 3 classes. It was justifiably concluded that the patterns obtained following hammer blows on the dry skull were similar to the findings in the living skull.

In the study of strain patterns in the human, dry and cadaver skulls were used. The effects of hammer blows on the midoccipital region near the parietal occipital suture, the midfrontal region, the anterior vertex at the midline, the lateral frontal, and the lateral posterior parietal regions were studied (3). Following midfrontal blows, the strain paths extended toward the roof of the orbit, along the orbital rim, and toward the frontosphenoidotemporal junction at either side. With a midoccipital blow there were evidences of extensive deformation about the foramen magnum. Lateral frontal and lateral posterior parietal blows caused cracks in the lacquer extending toward the temple; with the former the direction of the cracks was downward and posteriorly, and with the latter downward and anteriorly.

It was found that the human and lower forms differed materially in their strain patterns. In the human the deformations detected by this technique were more localized. However, the value of the experimental animal in establishing correlation of results in the animal under anesthesia, in the skull of the dead animal with contents intact, and in its dry skull, was of inestimable value. It could be definitely stated that the patterns ob-

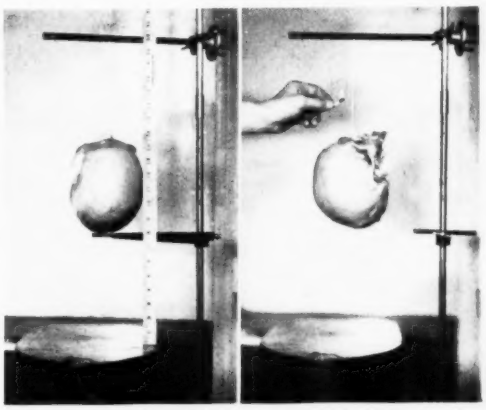

Fig. 1. This figure illustrates the method used for delivering known quantities of energy in certain locations in the skull. Before each experiment the skull is carefully weighed. It is then held at the proper distance from the steel plate to produce the desired energy at impact. The thread holding the skull is burnt with a match and following the impact the skull is caught on the rebound in order to avoid the possibility of secondary patterns from more bounces.

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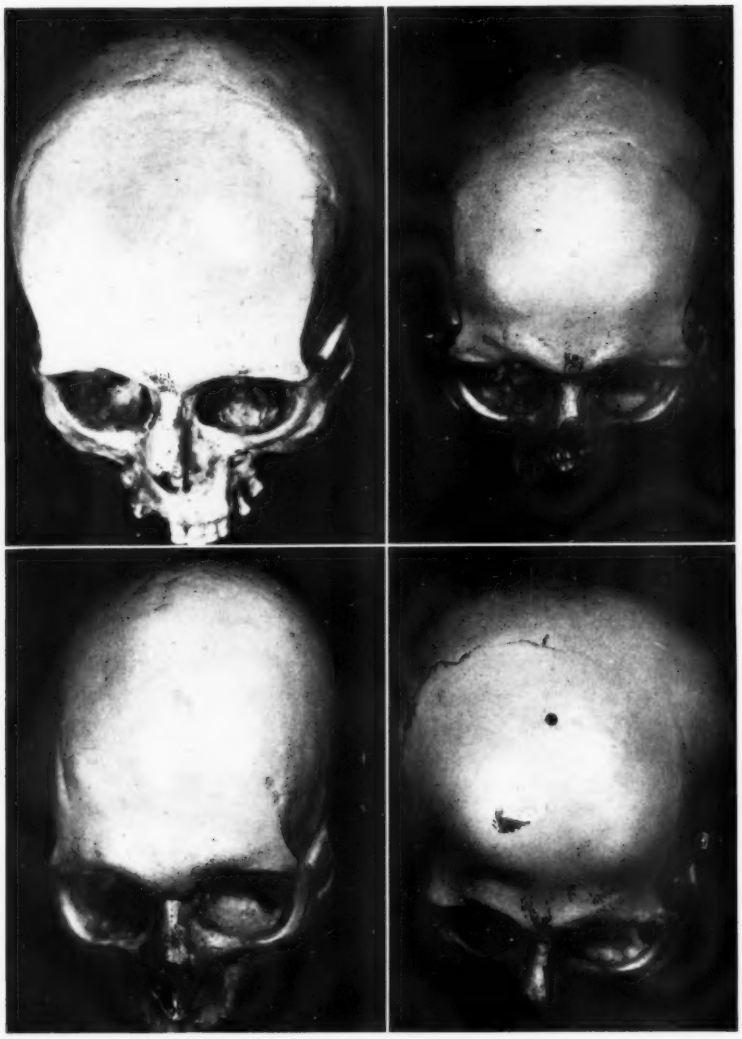

Fig. 2. The effect of 14.3 inch pounds of energy on the mid-forehead. Upper left is skull G with sensitivity of lacquer at 0.00105 inch per inch. Upper right is skull C with sensitivity of lacquer at 0.00095. Lower left is skull B with sensitivity of lacquer at 0.00095. Lower right is skull D with sensitivity of lacquer at 0.00095. Note that there are minimal deformation patterns in each instance with the exception of skull B which shows no effects from the blow.

tained in the dry skulls were essentially the same as in living animals under anesthesia. This does not signify that the magnitude of the strains is the same, but that the strain paths are similar. It was also deduced with reason that the effect of the skin and muscles on the strain pattern is negligible.

In the present paper certain quantitative data are given. The effects of a given amount of absorbed energy in various portions of the skull are

Fig. 3. Anterior vertex (midline) blow showing lines of strain extending toward the temple on both sides. This is skull B with sensitivity of lacquer at 0.00105 and with a blow of 14.3 inch pounds of energy.

described. Deformations resulting from blows of varying energy are also discussed.

PROCEDURE

Previous tests have shown that suture lines in dry skulls tend to interrupt the propagation of the cracks in the "stresscoat" lacquer. It was therefore decided to keep the skulls moist by immersing in normal saline solution between tests to prevent the disintegration of the tissue at the suture lines. One skull was permitted to remain dry as the tests progressed to determine any changes in deformation patterns resulting from the drying of the skull. Six human skulls, freshly obtained from the anatomical laboratories, and dissected free of soft tissues were used. Five were kept in normal saline solution, except when in use for strain determinations.

The skulls were removed from the saline solution in which they were stored several hours before coating in order to permit the excess moisture to drain off and evaporate. Just prior to spraying they were washed with ether. In no case was there any difficulty in getting the lacquer to adhere properly to the bone. The skull was first sprayed with a thin uniform undercoating of aluminum lacquer and after 15 minutes of drying a uniform coating of "stresscoat" lacquer was applied with a sprayer. Numerous calibration strips were coated at the same time. The lacquer was allowed to dry for 24 hours before a blow was administered.

In order that the results of tests made at different times could be correlated it was necessary to have the strain sensitivity of the lacquer within certain limits for all experiments. The range chosen was 0.00100 $\pm$ 0.00005 inch per inch of strain to produce cracks in the lacquer. When the test was about to be made, one of the calibration strips was tried to check the strain sensitivity of the lacquer for the existing temperature and humidity condition of the room. If the sensitivity was too great, the room temperature was increased until a calibration strip test showed that the sensitivity was 0.00100 $\pm$ 0.00005. The skulls were then tested. A room with constant temperature and humidity condition would obviate some of the above described operations. However, with

Fig. 4. Anterior vertex (midline) blow in skull E with sensitivity of lacquer at 0.00105 and a blow of 14.3 inch pounds. Note that the pattern in this skull is somewhat more extensive than in skull B, Fig. 3.

the use of the calibration strips an accurate and quantitative determination can be obtained.

The skulls weighed from 1.07 to 2.23 pounds. They were dropped on to a polished steel block weighing several hundred pounds. Therefore the energy absorbed by the steel block was negligible. The amount of energy absorbed by each skull was determined by its weight and the distance through which it was dropped. The velocities at the instant of impact varied from 5.4 to 9.9 feet per second. Each skull was supported so that the blow was delivered perpendicular to a plane tangent to the point of impact.

A series of tests were made on the six human skulls with blows delivered to the midfrontal, midoccipital, lateral frontal, lateral posterior parietal, and anterior vertex (midline) regions. The skulls were suspended above the steel block with thread. For occipital blows the thread was tied to the bridge of the nose; for frontal blows, to a hook in the foramen magnum; for lateral posterior parietal blows, it was tied to the zygomatic arch; for vertex blows, the skull was supported by a thread tied to a hook in the foramen magnum, and for the lateral frontal blow it was supported from a thread in the posterior parietal region tied to another tightly encircling the skull. The skull was then adjusted to the correct height above the steel block. The thread was burned, allowing the skull to drop on the steel block and it was caught on the rebound to prevent the formation of secondary strain lines in the lacquer due to additional bounces (Fig. 1).

The strain lines were next developed by coating the lacquered skull with red dye etchant. The excess etchant was removed with an emulsifier solution, leaving the dye only in the cracks. For purposes of photography each crack was then outlined with India ink. Faint cracks were gone over more lightly than heavy cracks which were stained with heavy lines of ink.

The formation of cracks in the lacquer indicated a minimum tensile strain of 0.00100 $\pm$ 0.0000 inch per inch. The magnitude of the strain in certain portions of the area of cracks may be considerably in excess of the value given above. For instance, in the region of a strain pattern the cracks are closely spaced at the center and more widely spaced toward the periphery. It is evident that a strain gradient exists with the strain of the

peripheral cracks being about 0.00100 inch per inch and increasing in magnitude toward the more closely packed cracks in the center.

In a series of experiments the same amount of energy (14.3 inch pounds) was used in different locations of the skull. Another group of experiments were carried out in which the quantity of energy applied to different regions was varied from 8 to 24 inch pounds (Table I).

EXPERIMENTAL DATA

In Table I the weight of each skull at the time of a given test is tabulated. Skull B was kept dry and the other 5 were immersed in normal saline solution between tests. Only skull C shows a marked change in weight. This was caused by the separation of a portion of the intracranial contents which remained attached to the internal surface of the skull during the first two tests. It should also be noted that in certain experiments the skull weight was higher than in certain previous tests. This is almost wholly due to the varying amount of aluminum and lacquer coating sprayed upon the skull. Both are heavy and can cause differences in weight depending upon the thickness of the layer applied to the surface of the skull. The strain patterns in the skull not immersed in saline (skull B) were essentially of the same distribution and direction as those obtained from the moist skulls, but as the tests progressed a separation of the sutures gradually occurred and the results obtained after the sixth test were discarded. Such a separation not only may prevent the propagation of strain paths but it constitutes an area of energy absorption, causing quantitative errors in results. This is evident from the examination of the strain patterns obtained with 14.3 and 20 inch pounds of energy, Tests B-2 and B-8. In the case of this skull the strain cracks were less extensive for the higher energy than for the lesser value. It is felt that the most reliable results are obtained in fresh skulls which are kept moist in saline between tests. However, skull D, which was kept moist throughout the tests, had a separation of the lambdoid suture on the ninth test and was then discarded. The experimental data given were completed during a period of 3 months.

A constant energy of 14.3 inch pounds was used in all skulls in the first 6 experiments (Table I). Varying amounts of energy were used beginning with test 7. The findings will be described under (1) blows of constant energy, and (2) blows of varying energy.

Results with constant energy. The application of 14.3 inch pounds of energy by the above-described procedure resulted in patterns of different magnitude in different locations. Following an occipital blow the deformations were more extensive. With the same amount of energy midfrontal blows resulted in very slight change. There was also some difference in magnitude of patterns obtained with blows at the same location but in different skulls. Such variations from skull to skull may be explained in part by differences in shape, contour, and thickness, and slight change in the location of the blow in successive tests. These data will be described in greater detail in the next few paragraphs.

Fig. 5. Lateral frontal blow in skull E with sensitivity of lacquer at 0.00105 and a blow of 14.3 inch pounds of energy.

Midfrontal blow with 14.3 inch pounds of energy. These experiments were carried out on 5 skulls kept in saline and 1 dry skull. In Figure 2 it is noted that minimal strain cracks are found in the frontal bones in 3 of 4 specimens. In 1 (skull B), a blow of 14.3 inch pounds of energy caused no cracks in the lacquer with a sensitivity of 0.00095.

Anterior vertex (midline) blow with 14.3 inch pounds of energy. A blow in the anterior vertex at

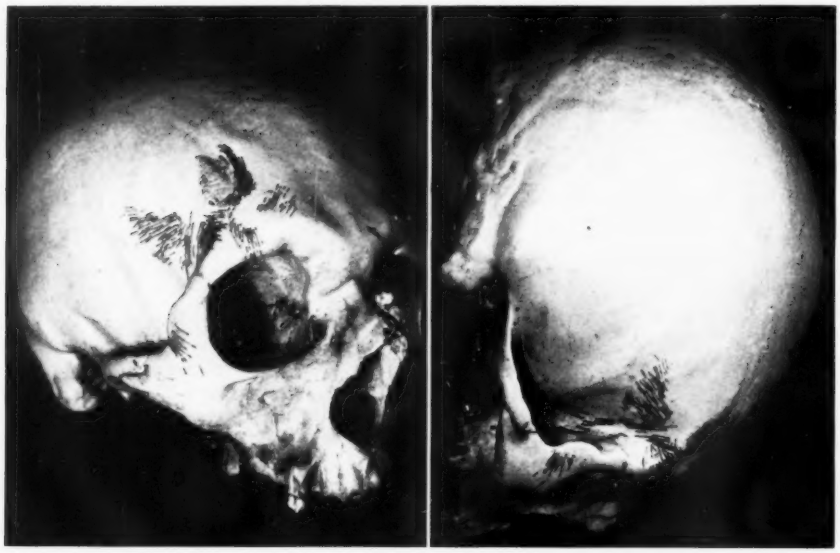

Fig. 6. Lateral frontal blow on the right side in skull B with 14.3 inch pounds of energy and sensitivity of lacquer at 0.00105. ‡In this experiment the skull fractured with line of fracture extending from the orbital rim to the middle of the area of impact.

TABLE I.— SKULL WEIGHTS AND DISTANCES DROPPED

Test No.*	Skull											
	B†		C		D		E		F		G	
	Weight	Distance dropped	Weight	Distance dropped	Weight	Distance dropped	Weight	Distance dropped	Weight	Distance dropped	Weight	Distance dropped
1	1.19	12	1.73	8 1/4	1.18	12 1/8	1.47	9 1/4	2.23	67/16	1.75	83/16
2	1.16	125/16	1.72	85/16	1.17	12 1/4	1.38	10 1/8	2.07	67/8	1.02	813/16
3	1.17	123/16	1.37‡	107/16	1.20	11 15/16	1.38	10 1/8	2.05	7	1.03	8 1/4
4	1.18	12 1/8	1.34	10 11/16	1.19	12	1.35	10 5/8	2.04	7	1.02	813/16
5	1.18	12 1/8	1.23	11 5/8	1.07	13 1/8	1.36	10 1/2	2.05	7	1.59	9
6	1.18	12 1/8	1.27	11 1/4	1.12	12 3/4	1.41	10 1/8	2.09	613/16	1.04	8 1/4
7	1.18	12 1/8	1.27	7 7/8	1.16	8 5/8	1.40	7 1/8	2.08	8 5/8	1.04	75/16
8	1.19	1015/16	1.25	6 3/8	1.16	07/8	1.36	14 11/16	2.08	8 5/8	1.02	12 5/8
9			1.31	123/16	1.17	13 11/16	1.39	73/16	2.08	5 5/8	1.05	611/16
10			1.29	14			1.43	14	2.11	9 1/2	1.05	149/16
11			1.28	15 5/8								
12			1.29	7 3/4								
13			1.32	183/16								

†Skull B, dry.
*Tests 1 through 6 constant energy tests (14.3 inch pounds); 7 through 13, variable energy tests.
‡The sudden decrease in the weight of the skull was due to separation of a portion of intracranial contents attached to the inner surface of the skull.
Note: Skull D, test D-9. On this test there was separation of lambdoid sutures.
Skull B, test B-6. On this test there was linear fracture of the skull in the right lateral frontal region.
Weights and distances in pounds and inches.

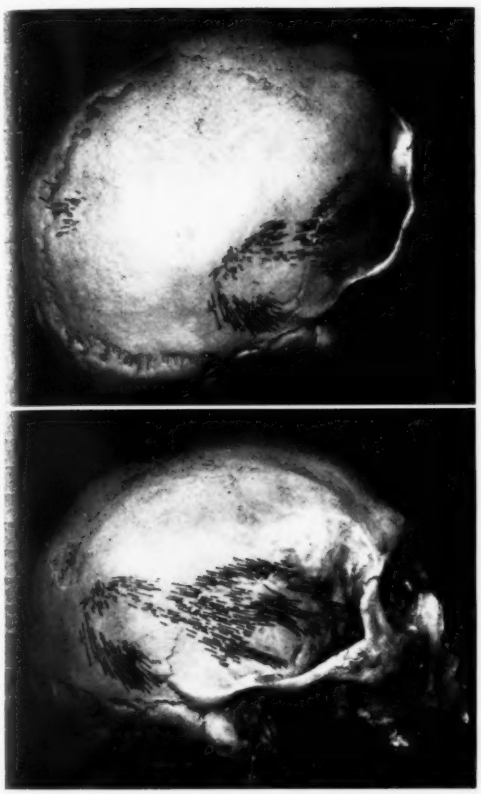

Fig. 7. Posterior parietal blow in skull E with 14.3 inch pounds of energy and sensitivity of lacquer at 0.00100 inch per inch. Note the extensive deformations toward the parietosphenoidofrontal junction.

the midline caused deformation patterns to appear along the lateral frontal and frontoparietal junction on both sides. There is some variation from skull to skull as concerns magnitude, but the general direction of the paths of strain are the same (Figs. 3 and 4).

Lateral frontal blow with 14.3 inch pounds of energy. A blow in the lateral frontal region caused deformation patterns to appear in the parietotemporal region and the frontosphenoidotemporal junction. The cracks extend down and posteriorly except near the zygomatic arch where their direction is downward and forward. In Figures 5 and 6 are shown the strain patterns following a lateral frontal blow with a sensitivity of the lacquer at 0.00105 in both instances. Note that in Figure 6 there are cracks in the lacquer covering the zygoma.

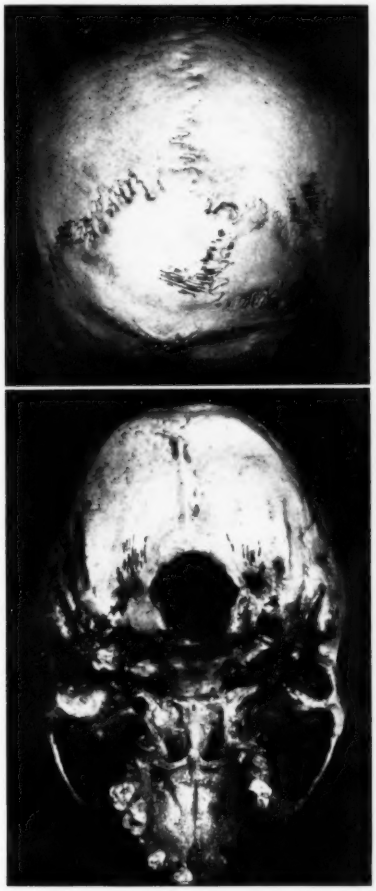

Fig. 8. Occipital blow at the midline with 14.3 inch pounds of energy in skull E. Note the patterns over the squamous portion of the occipital and parietal bones. Note also the pattern at the base of the skull about the foramen magnum.

Lateral posterior parietal blow with 14.3 inch pounds of energy. Extensive deformations occurred with such blows. The paths of strain were downward and toward the "temple" (Fig. 7).

Midoccipital blow with 14.3 inch pounds of energy. A blow on the squamous portion of the occipital bone near the lambdoid suture at the midline caused some local deformations and deformations about the base of the skull at the foramen magnum (Fig. 8). In Figures 8 and 9 are seen differences in the magnitude of these strains, but the direction of the paths are the same. This may be in part explained by differences in skull

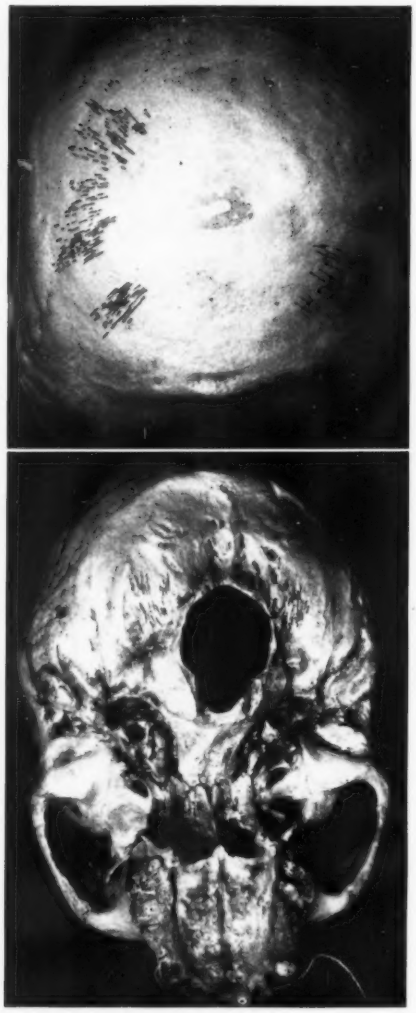

Fig. 9. Occipital blow in skull B with 14.3 inch pounds of energy. Sensitivity of lacquer at 0.00100. Extensive deformations are noted about the blow and at the base of the skull. This variation from skull to skull as concerns magnitude is seen frequently, but it should be noted that the distribution and direction of the patterns are essentially the same.

thickness and contour as well as by a slight change in the location of the blow from skull to skull. In one skull, "contrecoup" deformations with resultant cracks in the lacquer were noted in the frontosphenoidotemporal region on one or both sides following midoccipital blows (Fig. 14).

TABLE II.—VARIABLE ENERGY TESTS

Energy in pounds	Blows		
	Occipital	Posterior parietal	Frontal
8	C-8		
	D-8		
10	C-7	C-12	
	D-7.	E-7	
	E-9		
	G-9		
12		F-9	
		G-7*	
14.3	B-2	C-2	C-3
	C-1	E-1	G-3
	D-1	F-1	B-3*
	E-2	G-1	D-3
	G-2		
16	C-9		
	D-9		
18		F-8	C-10
20	B-8	F-10	C-11
	E-10	E-8	G-8
24			C-13 G-10

*No cracks.
Capital letters identify skulls and numerals following the letters signify test number.
Note: Tests B-7 and F-7 (Table I) in locations other than above shown, hence not included in this table.

Results with varying energies. Energies varying from 8 to 24 inch pounds were used in the midfrontal, midoccipital, and lateral posterior parietal regions of the skull. In order to obtain more reliable information varying energies were used in different locations on the same skull. These procedures were carried out on three specimens, and comparison of results lead to similar conclusions. Whereas in the occipital region an absorbed energy of 8 inch pounds causes threshold deformations, in the midfrontal region 14 to 18 inch pounds of energy is necessary to obtain beginning cracks in the lacquer. Eight inch pounds caused no deformations of sufficient magnitude to crack the lacquer in lateral posterior parietal blows, but threshold deformations were obtained with the use of 10 inch pounds. The illustrations for this portion of the work are photographs of the same skull struck with blows of varying energy (skull C, Table I).

Midfrontal blow with varying energies. In all skulls energies of 10 and 12 inch pounds caused no cracks in the lacquer. With 14.3 inch pounds the

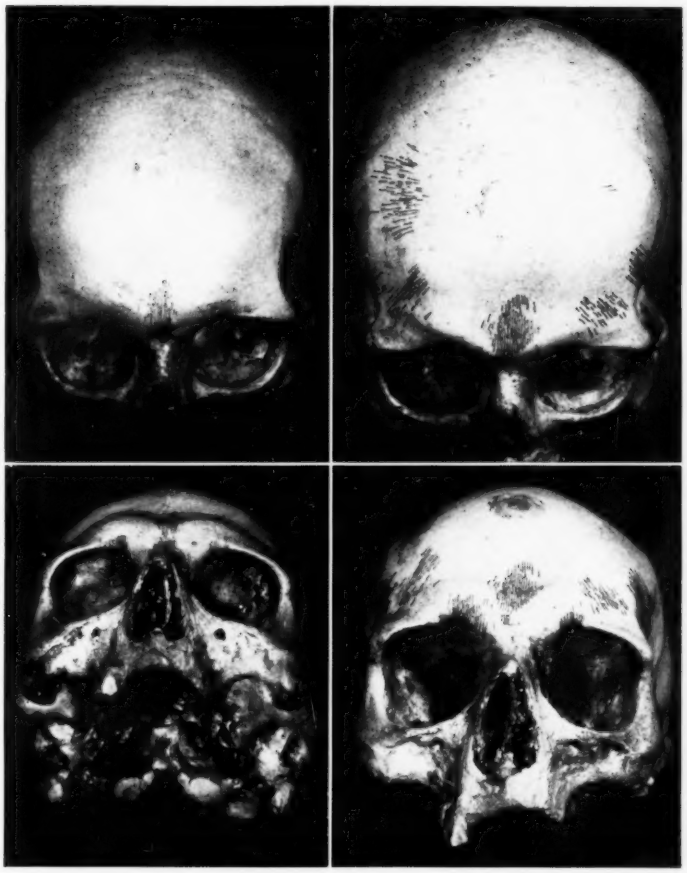

Fig. 10. A midfrontal blow in skull C with 20 inch pounds of energy (left upper and lower); with 24 inch pounds of energy (right upper and lower). Note the increasing intensity of deformation patterns with increase in the injuring energy. Sensitivity of lacquer 0.00105 in both experiments.

results are shown in Figure 2. In Figure 10 are shown photographs of deformation patterns following the use of 20 and 24 inch pounds of energy. In Figure 11 are seen the lateral views of these skulls. With 20 inch pounds of energy, only faint and minor deformations are noted. With 24 inch pounds, the cracks are more evident, heavier, and more numerous and comparable to those obtained in the midoccipital blow with 14.3 inch pounds of energy.

Midoccipital blow with varying energies. Patterns were obtained with as little as 8 inch pounds of energy (the sensitivity of the lacquer at 0.00095). With 10 inch pounds of energy the deformations are more extensive. With 14.3 inch pounds of energy the deformations at the base are more numerous and there are some cracks in the lacquer covering the parieto-occipital region. With 16 inch pounds of energy, the cracks in the lacquer extend to the base of the middle fossa, to the petrous bones and the zygomatic arches. The cracks are heavier, longer, and more numerous (Figs. 12 and 13). In Figure 14 are shown contrecoup deformations of sufficient magnitude to cause cracks in the lacquer at the frontosphenoidotemporal junction. They were first seen with 10 inch pounds of energy and are more extensive with blows of 14.3 and 16 inch pounds.

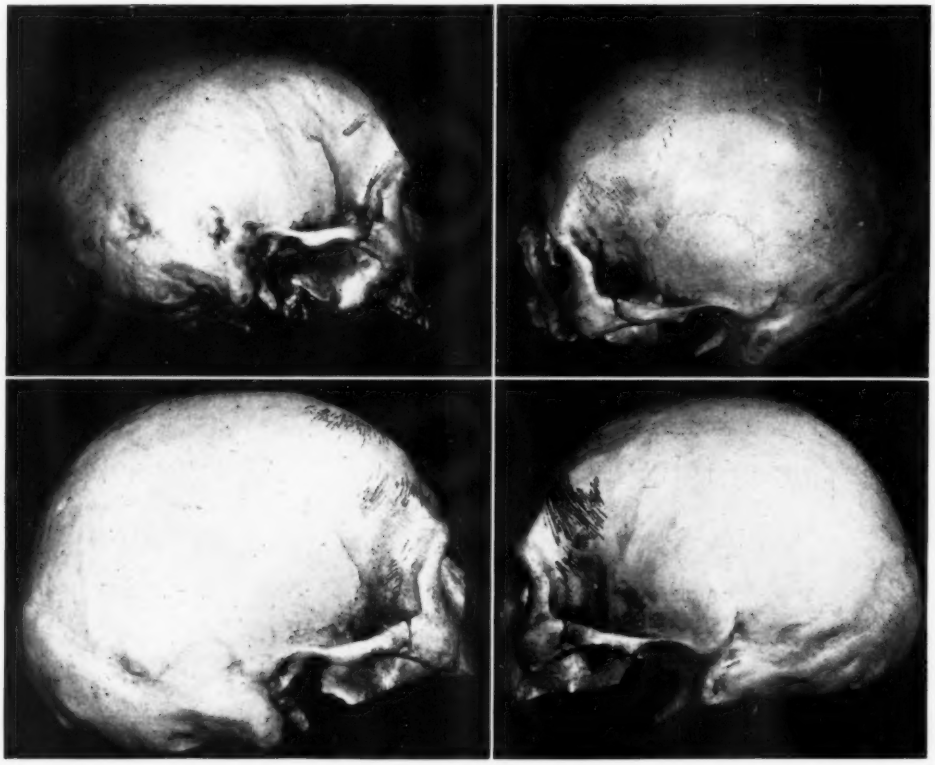

Fig. 11. Lateral view of skull C in experiments described in Figure 10. The upper two photographs show the results of a blow to the midfrontal region with 20 inch pounds of energy and the lower two photographs are the findings following a blow with 24 inch pounds of energy. Sensitivity of lacquer 0.00105 in both experiments.

With 8 inch pounds no contrecoup cracks were noted.

Lateral posterior parietal blow with varying energies. Lateral posterior parietal blows with 10 inch pounds of energy caused a few faint lines of deformation in the parietotemporal region. With 14.3 inch pounds the deformations were much more extensive as shown in Figure 15.

EVALUATION OF STUDY

The human skull is well suited for study with the "stresscoat" technique. After convincing experimental data (3) it was concluded that deformation patterns in the dry skull are the same as in the living. The influence of scalp and muscles on the strain path is negligible. It should be emphasized that this method shows only tensile strain. Compression strain cannot be visualized. It should also be noted that only the behavior of the external surface of the skull has been studied thus far. Tensile strains of the internal surface are doubtless important following certain severe blows. Strain patterns may vary depending upon the location of the blow and small changes in location may produce altogether different paths of deformation. More work is needed to learn in greater detail the behavior of the skull to blows of varying location.

The strain paths depend upon the shape, contour, and thickness of the skull. Certain "chance" differences in skull thickness may produce profound differences in patterns. In our experiments hammer and deceleration blows have been used. Actually, in automobile accidents and falls there may be a combination of energies operating on the skull in succession or simultaneously in more than one location. Depending upon the velocity of the blow, deformation patterns may be given a certain

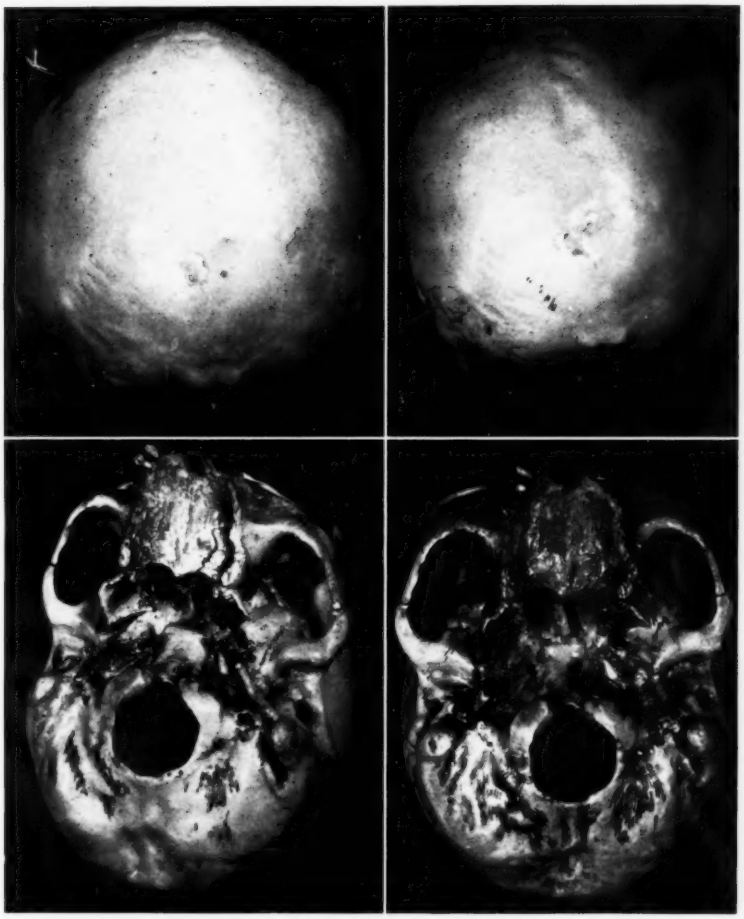

Fig. 12. Midoccipital blow with graduated energies on skull C. To the left, results with 8 inch pounds of energy and sensitivity of lacquer at 0.00105; to the right, results of a blow with 10 inch pounds of energy with sensitivity of lacquer at 0.00105.

directional quality. High velocity energies may cause localized deformation with comminution, depression, or perforation of bone. At the present time, we have no information concerning the behavior of a skull with energies of high velocity. In the present study velocities at the instant of impact varied between 5.4 and 9.9 feet per second.

In general, the paths of strain brought out by these studies parallel fracture lines seen clinically in the human. The deformation patterns are more common in the weaker than in the buttressed zones of the skull (4). Thus, very few cracks are seen in the lacquer extending longitudinally along the median plane of the frontal and occipital bones. Relatively few deformation patterns are seen along the zygomaticofrontal and the petrosoparietal buttresses. Clinical data corroborate these findings. Fractures resulting from general deformations of the skull in general follow the strain paths seen in these experiments. With blows in the posterior parietal region, a fracture line extending down and forward toward the temple is commonly seen. Fracture lines in the occipital bone in general follow the strain paths seen in these tests. The three types of fracture seen include linear fractures extending into the

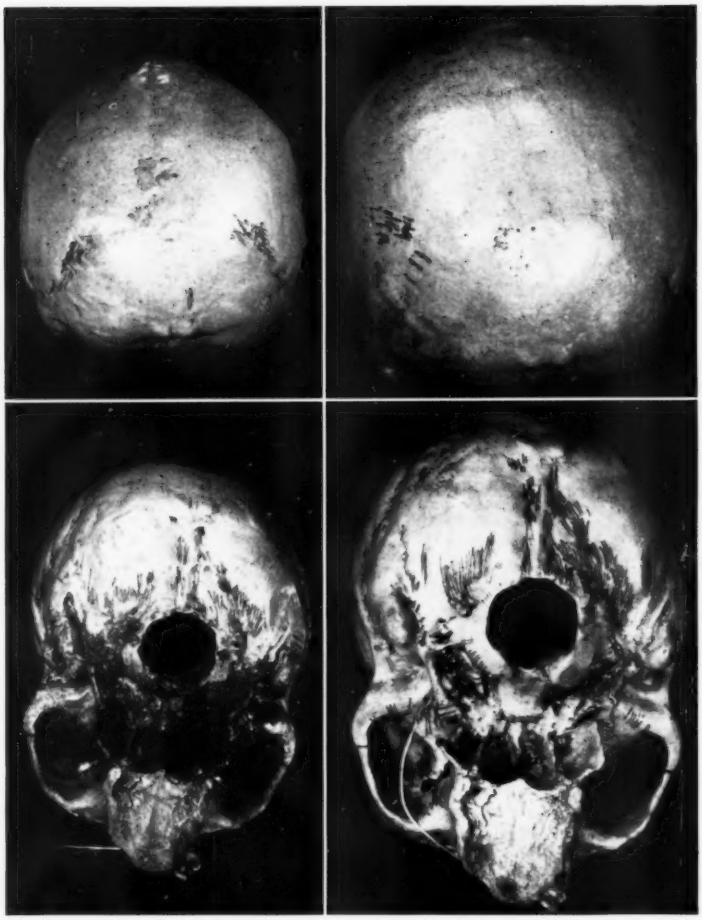

Fig. 13. To the left, an occipital blow with 14.3 inch pounds of energy and sensitivity of lacquer at 0.00105; to the right, an occipital blow with 16 inch pounds of energy and sensitivity of lacquer at 0.00005 inch per inch (skull C).

foramen magnum, linear fractures extending lateral to the foramen, and fractures which extend down toward one or the other of the large openings at the base of the skull such as the jugular foramen or the hypoglossal foramen. These may then extend forward toward the petrous bone. Fracture lines in frontal blows are usually seen extending toward the roof of the orbit in a paramedian position, much in the same direction as in the strain paths with midfrontal blows. It is obvious that with high velocity energies there may be localized deformation with comminution, depression or perforation. It has also been mentioned previously that depending upon the size, shape, and velocity of the injuring object, a certain direction may be given the strain paths.

Certain threshold values may be deduced from a study of Table II. Whereas in the occipital region 8 inch pounds of energy are sufficient to cause deformation patterns in the lacquer of the sensitivity used, in the midfrontal region 14 to 18 inch pounds of energy are needed for deformations of similar magnitude. In the parietotemporal region, threshold values are obtained with 10 inch pounds. It is therefore apparent that relatively minor blows may result in deformation

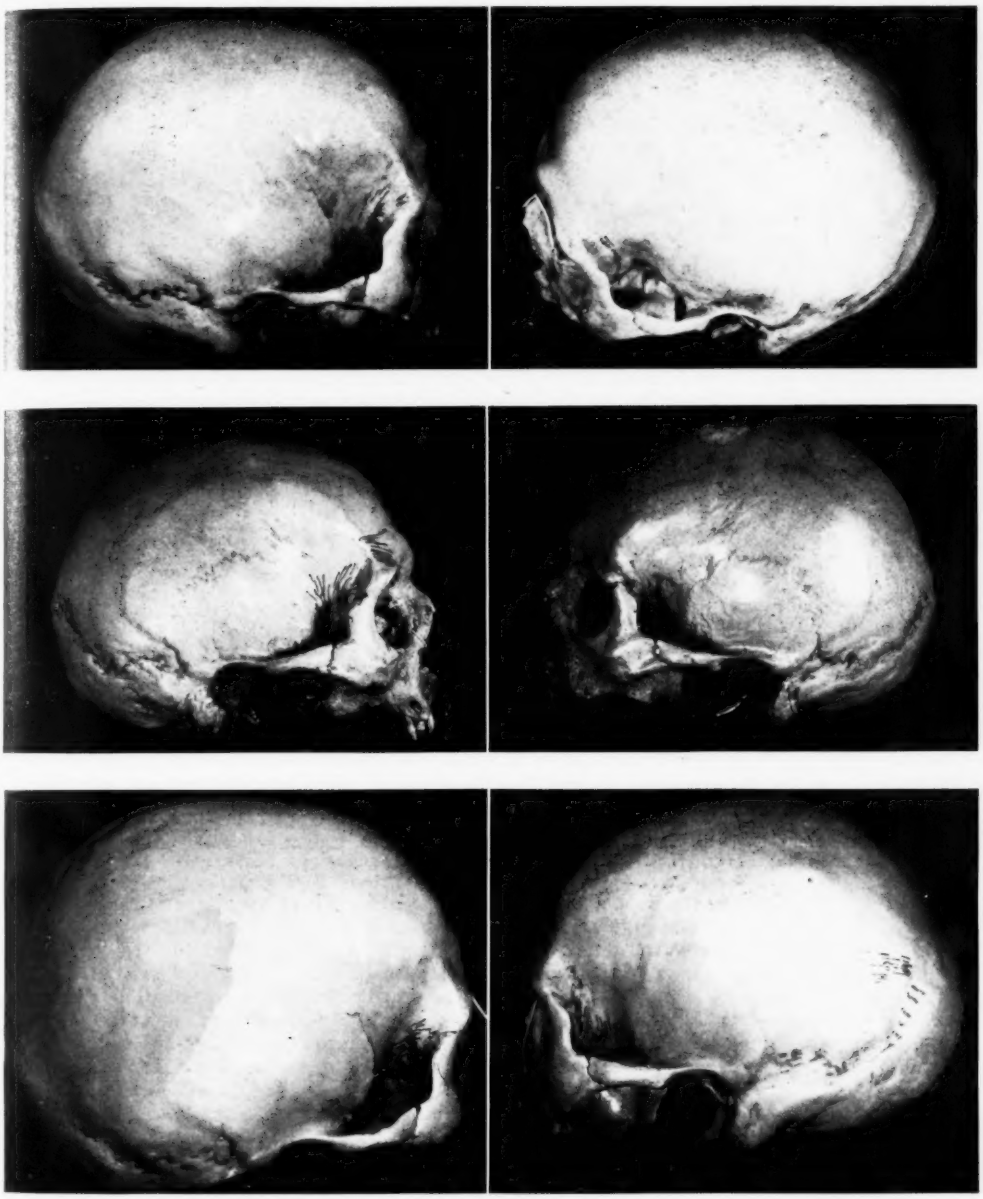

Fig. 14. Lateral view of skull C with midoccipital blow of 10 inch pounds of energy, 14.3 inch pounds of energy and 16 inch pounds of energy. Note that with 10 inch pounds (upper row) deformations are only slight; with 14.3 inch pounds they are more extensive (middle row), and they are most widespread with 16 inch pounds of energy (lower row). The deformation patterns are found at the frontosphenoidotemporal junction on one or both sides (contrecoup deformations).

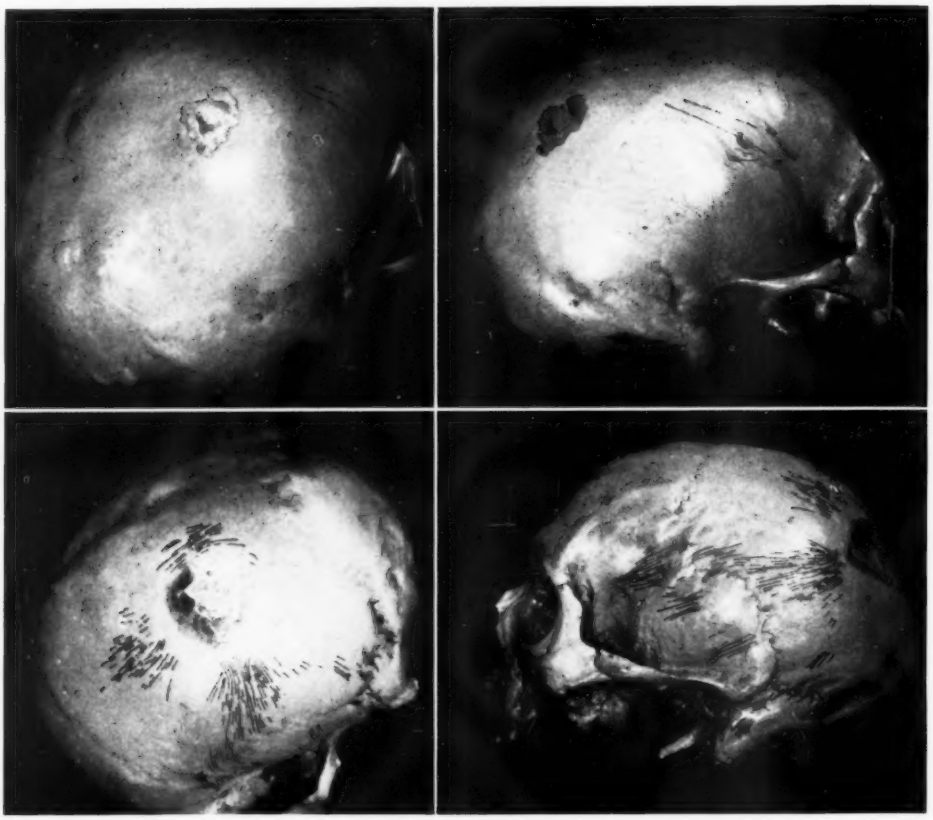

Fig. 15. Lateral posterior parietal blow in skull C with 10 inch pounds of energy (upper row), and 14.3 inch pounds of energy (lower row). The sensitivity of the lacquer 0.00095 and 0.00100, respectively.

of the skull with failure in certain regions. The ease with which deformations are produced in the region of the foramen magnum and parietotemporal regions may explain how in certain injuries linear fractures may obtain in these locations without loss of consciousness. A small amount of energy may be dissipated in causing deformation and failure of the skull. However, it may not be of sufficient magnitude also to result in adequate pressure waves in the cranial cavity to cause posttraumatic unconsciousness. A severe blow in the occipital region is associated with extensive deformation at the base of the skull thus helping to cause intracranial pressure waves to add to the serious intracranial injury. Whereas a weak blow may cause a symptomless fracture line, a severe blow may result in a serious state. It is clinically known that patients with fracture of the base may be seriously ill and yet there are instances in which the symptomatology is very slight.

In one skull (C), occipital blows were consistently associated with deformation patterns at the frontosphenoidotemporal junction on one or both sides. From their position, they may well be called contrecoup deformations. Contrecoup fractures may thus be possible. Clinically, the association in some cases of "black eyes" with a fall resulting in an occipital blow may be explained by contrecoup deformations in the region of the origin of the temporalis muscles with post palpebral bleeding.

SUMMARY

1. The amount of absorbed energy necessary to cause threshold deformations is different in vari

ous parts of the skull. In the midfrontal region blows of 14 to 18 inch pounds of energy cause deformations of a magnitude similar to those obtained with 8 inch pounds in midoccipital blows.

2. A small amount of energy may be dissipated in deformation and failure (fracture) of the skull. However, it may not be of sufficient magnitude also to result in adequate pressure waves in the cranial cavity to cause posttraumatic unconsciousness. This may explain how in certain locations of the skull linear fracture may obtain without unconsciousness.

3. Contrecoup deformations of the frontosphenoidotemporal junction following midoccipital blows were obtained in one out of six skulls. This argues in favor of contrecoup fractures in certain cases.

4. The distribution and direction of deformation patterns generally parallel fracture lines in clinical cases. However, the shape and velocity of the injuring object may influence and give strain paths a directional quality.

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SPONTANEOUS RUPTURE OF THE ESOPHAGUS

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SPONTANEOUS rupture of a presumably normal esophagus has been reported only infrequently. The occurrence, therefore, of 3 cases in a period of 1 year in the experience of the senior author has prompted us to report these cases and review the literature.

The earliest case recorded is one by the Dutch physician Boerhaave, in 1724 (12). His brilliant description over 200 years ago of the onset and course of the disease and of the findings at autopsy leave relatively little to be added today. A few lines with regard to this report therefore are of interest:

Quoted from van Swieten: "We have a surprising observation given us by the celebrated Boerhaave which is, perhaps the only one published, namely, the illustrious Baron Wassenaer, Lord High Admiral to the Republick, after intense straining in vomiting broke asunder the tube of the esophagus, near the diaphragm, so that after the most excruciating pains the aliments which he swallowed passed, together with the air, into the cavity of the thorax, and he expired in twenty four hours."

Quoted from Liebetaud: "From him (Boerhaave) it appears that the Admiral, who was more than fifty years old, suffered an annoyance at the pit of the stomach three days after feasting sumptuously. During the efforts to obtain relief by vomiting, the sudden pain, 'as from some rupture or tearing' occurred. During the progress of the affection he was constantly tortured with extreme pains. In the meantime increased suffering threatened syncope; there was neither fever, cough nor difficult breathing. The vehemence of the pains prevented any motion of the body and there was scarcely any remission from their great severity. They seemed to be seated primarily within the chest near the diaphragm, thence extended to the back and throughout the entire chest. Finally the strength gave way and he died suffocated, the horrid symptoms continuing. Nothing noteworthy was found in the abdomen; a large amount of gas escaped from the first incision into the pleural cavity. The lungs and heart were unaffected. A marked and unusual smell came from the chest, as if proceeding from some putrid fluid in which the posterior parts of both lungs were bathed; this fluid, amounting to six pounds, did not differ in the least from the contents of the stomach. The body having been thoroughly cleansed, there was found a transverse rupture through the esophagus near the diaphragm."

The first review of this subject in the American literature was by Reginald Fitz of Boston in 1877 (12). He recorded a case of his own and described the disease at some length. The next comprehensive review appeared in 1914 (55). At that time Walker was able to find only 22 cases recorded in which rupture took place spontaneously in an esophagus otherwise normal. His case is the first of the 4 reported in the literature which have been diagnosed before death (1, 16, 50, 55). Since Walker's review several articles on this subject have appeared (1, 8, 16, 19, 32, 41, 50). Most have been solitary case reports. However, Gott in 1933 in a 10 year review of all admissions to the Louisville City Hospital was able to find 4 cases. Ridgway and Duncan in their review of 1937, believe that 3 of these cases are not representative of true spontaneous rupture. The latest review by Klein and Grossman in 1943 included only 40 cases gathered from the literature. We have been able to find only 1 case report since then (8). In contrast to rupture, perforation of the esophagus is not uncommon. It may follow the ingestion of a foreign body (3, 5, 18, 28, 30), instrumentation (21, 23, 25, 26, 53), external trauma (31) or erosion of a carcinoma, or result from a stricture (54) or syphilis (15). This is not the sort of injury under consideration, however.

Spontaneous rupture of the esophagus occurs at the lower end—usually within an inch or two of the cardia. At times the rupture extends down into the cardiac portion of the stomach. The posterior or posterolateral wall usually on the left side is the site most commonly involved. The rupture is usually a solitary one and in only 2 of Walker's 22 cases failed to be a longitudinal slit-like opening. The fissure may be one to several centimeters in length. Though in some cases there is evidence of pre-existing ulceration usually the mucosa and muscular coats are quite normal except for the immediate site of rupture.

Congenital weakness of the area seems unlikely as an explanation for the rupture. Twenty-five of the 35 cases reported by Ridgway and Duncan occurred during the third, fourth, and fifth decades of life. Menne and Moore could find only 3

cases in childhood—a time when congenital abnormalities are likely to become manifest and when vomiting is especially prone to occur. Also the reported cases usually do not present thinning of the muscular coats.

Ulcerative esophagitis and peptic ulcer of the esophagus are entities which are now recognized and perforation and hemorrhage can occur in these ulcers (4, 13, 27, 38). It is questionable whether such cases should be included in a consideration of spontaneous rupture of the esophagus. The possibility of an acute linear peptic ulceration in the remaining cases still is present. Moreover the factor of peptic digestion of a traumatic fissure is even more difficult to rule out. Two cases have been reported of rupture of the esophagus after extensive burns (49, 51). In 1 Curling's ulcers were present also in stomach and duodenum (51).

There seems little question that the most important factor in spontaneous rupture of the esophagus is that of sudden increase in pressure. Cases have been recorded following crushing injuries of the abdomen, (1, 41) during convulsive seizures (32), and during defecation (42). In 23 of the 35 cases reported by Ridgway and Duncan vomiting had preceded the rupture. Of the 25 in whom an accurate history could be obtained, 18 were alcoholics. Though the alcohol in itself may have a deleterious effect on the esophagus, it seems likely that the nausea, retching, and vomiting associated with excessive drinking are the more important factors in rupture.

In 1884 (8) McKenzie demonstrated experimentally that the lower end of the esophagus is the weakest portion. Weiss and Mallory (37, 56) were able experimentally to reproduce linear fissures of the lower end of the esophagus and cardia by increased pressure.

Weiss postulated the following (20): Vomiting is a complicated and highly co-ordinated mechanism. The pylorus closes and the cardia and esophagus dilate. A wave of counter peristalsis is set up, and the esophagus relaxes. A sudden convulsive rise in intra-abdominal and intragastric pressure empties the stomach. If vomiting is repeated the vomiting center becomes fatigued. Co-ordination is lost and the esophagus and diaphragm fail to relax in the face of the sudden increase in pressure, resulting in rupture. The fact that at least 2 cases (12, 54) occurred during strenuous vomiting in attempts to dislodge a bolus of food in the esophagus would lend support to this theory. Rupture has been reported in association with neurogenic disturbances but this is certainly uncommon and an unimportant and irrelevant factor in this presentation (9, 39).

In only 5 of the 35 cases reported by Ridgway and Duncan were the patients females. The significance of this is not certain unless the picture of alcoholism is considered predominant in the male.

Rupture of the esophagus usually takes place during an episode of vomiting, often precipitated by a bout of drinking or a heavy meal. The patient experiences sudden severe pain high in the upper abdomen or epigastrium or beneath the sternum. Frequently the pain radiates to the back or to the interscapular region—the left side being more often the site of reference than the right. With the onset of the pain, some have felt a sensation of something tearing or giving way in the chest. If vomiting is repeated the vomitus is likely to be bloody. Though the pain is usually severe and persistent and often is not relieved significantly by morphine, in some cases the pain has not been severe but has been more in the nature of a pressure or discomfort in the region of the rupture. Food particles are often found in the posterior mediastinum. Rupture into the pleural cavity (usually the left) is not uncommon. If this be true gastric contents may be found here also. Even though the pleural cavity is not actually invaded, pleural effusion is often present because of the adjacent mediastinal irritation. Though hemorrhage may be rather profuse after rupture the real threat to the patient is that of a fulminating mediastinitis and death. There has been no case reported so far in the literature in which recovery has taken place, though in an editorial footnote in the 1944 *Yearbook of Surgery*, Graham mentions 2 cases which resulted in empyema and recovery following drainage of the empyema. This same course has been reported following perforation of the esophagus.

On examination the patient is found to be obviously critically ill. Shock is profound. Thirst is extreme and the patient may be able to take fluids by mouth with little difficulty or discomfort, though some discomfort may be felt locally at the site of rupture as the material swallowed passes over it. The blood pressure is low, the body temperature may be depressed and the pulse is rapid. The skin is cold and clammy; there is a pallor which quickly gives way to marked cyanosis. Dyspnea is often present and the respiratory rate is increased.

Initially findings on examination of the chest may be normal, and the upper abdomen is somewhat tender and guarded. However, as time passes the abdominal signs tend to diminish and the chest signs become more prominent with the development of râles and dullness at the lung bases. Some have been able to detect an abnormal

sound of fluid and air in auscultation of the chest during the swallowing of water. In about half the cases subcutaneous emphysema eventually develops. It is manifested first in the suprascapular fossae and the suprasternal notch but later extends to involve the entire neck, face, trunk, and even the extremities. Wangensteen once said: "In the drowsy small hours of the night, I have once made the diagnosis of spontaneous perforation of the lower esophagus over the telephone in conversation with the surgical resident on the basis of subcutaneous emphysema in a patient who had upper abdominal pain, no dyspnea, or cough." (45). If rupture extends into the pleural cavity physical signs of hydropneumothorax develop and gastric contents may be aspirated from the chest. Three cases diagnosed before death were discovered in this way (1, 16, 50). X-ray examination usually shows mediastinal emphysema within a few minutes of the time of rupture, long before the air becomes evident in the subcutaneous tissues of the neck. A fluid level in the mediastinum may also be demonstrated by x-ray examination.

The differential diagnosis includes coronary occlusion, dissecting aneurysm of the aorta, pulmonary embolism, spontaneous pneumothorax, ruptured peptic ulcer, acute pancreatitis, and mesenteric occlusion. The abdominal pain is so prominent a feature that many have been subjected to exploratory laparotomy (1, 24, 52, 55).

CASE REPORTS

Case 1. L. B. H. This 55 year old white man, was admitted September 18, 1944. He was a known alcoholic for many years and had recently been on a prolonged spree. The day of onset of his illness he did not feel well but had no particular complaints until 6:00 p.m. when he suddenly vomited before dinner. Some blood was seen in the vomitus. There was some upper abdominal pain and substernal distress but no rigidity of the abdomen and little tenderness. His pain increased and the upper abdomen became rigid. In addition a tightness of the lower chest was noted. The diagnosis of ruptured gastric or duodenal ulcer or acute pancreatitis was considered so he was explored 6 hours after the onset of his trouble. Nothing was found. He continued to vomit bloody material and gastric suction drainage was instituted. In spite of supportive treatment he failed to do well and showed marked circulatory collapse. The senior author was called in consultation the following morning. At this time subcutaneous emphysema was found on both sides of the neck. A chest film showed air in the mediastinum. An electrocardiographic tracing was normal. The serum amylase was 75. A diagnosis of rupture of the esophagus was made. The patient was *in extremis* at the time, however, and nothing could be done. He died at 6:00 p.m. that night, 24 hours after the onset of his illness.

At autopsy there was found a longitudinal rent 2 centimeters in length at the lower end of the esophagus. The surrounding mediastinal tissues up to the bifurcation of the trachea were inflamed, discolored, and necrotic. The right pleural cavity contained 450 cubic centimeters of clear amber fluid and the left 600 cubic centimeters with much admixture of blood on this side. The mediastinal borders of both lungs adjacent to the area of rupture showed fibrinous exudate. The stomach and duodenum were normal. However, blood was present in the intestinal tract.

Case 2. No. 42030. This 70 year old white man was admitted to the Hospital of the University of Pennsylvania on October 10, 1944, on the service of Dr. Edward Rose and Dr. William H. Erb. In 1930 a resection of the sigmoid was carried out because of a nonspecific infectious granuloma. A fecal fistula had persisted following the resection. For a few days prior to the present admission he had suffered some nausea. At 10:00 a.m. on the day of admission he had a sudden onset of vomiting. A second bout of vomiting contained blood. In a few minutes excruciating pain in the left lower chest and back developed which could not be relieved by morphine. The chest was clear on admission. His white blood count was 10,000. A survey film of the abdomen showed only large bowel distention. A chest film was negative. The electrocardiographic tracing was equivocal. He went into profound circulatory collapse and died 19 hours after the onset of his illness.

The clinical diagnoses included: coronary occlusion, dissecting aneurysm, and mesenteric thrombosis. Rupture of the esophagus was also considered when the senior author was called in consultation since Case 1 had been seen by him only the month before.

At autopsy the right pleural cavity was free of fluid. The left side contained air under pressure and 1500 cubic centimeters of brownish red fluid with clots. There was rupture of the mediastinal pleura in the region of the esophageal hiatus, and the lung was collapsed. A longitudinal rupture of the esophagus was found, 3 centimeters in length at the esophagogastric junction with no evidence of pre-existing ulceration. Two benign prepyloric ulcers were found in the stomach.

Case 3. E. Y., No. 65235. This 44 year old white woman was admitted on August 15, 1945, to the senior author's service at the Hospital of the University of Pennsylvania with a history of abdominal pain of 30 hours' duration, requiring morphine for relief. Though there had been some nausea and vomiting these were not prominent features in the history. On examination she was found to have a tender mass in the left lower quadrant, also felt on pelvic examination. A diagnosis of strangulation was made and exploration was carried out the day of admission. A gangrenous loop of ileum was found. Resection and primary anastomosis were performed. She did very well for 24 hours after operation. Her abdomen was soft and flat and peristalsis was present. Suddenly she developed a heavy substernal pressure with palpitation and generalized weakness and a sensation of smothering. She was pale and had a marked tachycardia. A medical consultation was obtained and an electrocardiographic tracing was done. Coronary occlusion could not be ruled out, but the consultant felt that pulmonary embolism was a more likely diagnosis. She was given morphine, oxygen, and plasma and seemed to rally for a time. Ten hours later, however, she again developed substernal distress, palpitation, and weakness. She went into increasingly profound peripheral circulatory collapse with intense cyanosis and cold clammy extremities. The pulse became imperceptible and the blood pressure unobtainable. A second electrocardiographic tracing was made and no changes could be found in it which were not consistent with profound circulatory failure. A Levine tube had been placed in the stomach following operation. The tube was removed at this time (35 hours after operation) because of respiratory distress. Repeated vomiting of coffee ground material followed removal of the tube, but it was not replaced because of fear of trauma to the bleeding site.

She failed to respond to treatment for shock and died, 30 hours after her first attack of substernal distress and 54 hours after operation.

At autopsy there was found a rupture of the anterior wall of the esophagus at the distal end. The rent was about 1.5 centimeters in diameter. The surrounding wall showed thinning of the muscularis. The adjacent mediastinal tissues were deeply stained with partially digested blood. The pleural cavities contained small amounts of straw colored fluid.

The possible rôle which the Levine tube may have played in this case is uncertain. Constant gastric suction drainage is now often used for periods of much longer than 24 hours with no difficulty at all. Of passing interest was the discovery at autopsy of an adenoma the size of a walnut in the cortex of the right adrenal (unsuspected).

Spontaneous rupture of the esophagus is no doubt much more common than appreciated. Many of the deaths in the past must have been erroneously attributed to the factors listed in the differential diagnosis. Postmortem examination is necessary to rule out the disease and has been the sole method of diagnosis in most of the cases reported. An autopsy, unless carefully conducted, may overlook the lesion. If the esophagus is divided just above the cardia and not removed *en bloc* with the stomach, the pathologic picture may be missed or destroyed.

Ninety per cent of perforations of the esophagus occur in the cervical region (22). For a long time the anatomy of the neck (7, 11, 33) and the methods of spread of infection in it (43, 45, 47) have been well understood. Many cases of perforation in this area have recovered following prompt anterior cervical mediastinotomy, even without the aid of chemotherapy (2, 6, 22, 29, 44, 46). This approach is of value for perforation or infection only above the level of the fourth or fifth dorsal vertebra—a level well above the area of the mediastinum involved in spontaneous rupture of the esophagus.

Posterior mediastinotomy was first described on the cadaver by Nassilow in 1888 (14). In 1898 Cavazzoni drained an abscess in the posterior mediastinum by the dorsal approach with recovery of the patient (14). The approach is now well understood and many patients have since been drained in this way with recovery (34, 35, 36). These have all been cases of a perforation recognized immediately or of a localized abscess. With the agents now available the question rises as to whether chemotherapy alone can replace operation in rupture or perforation of the esophagus as in the case reported by Heatly. It seems likely that extensive rupture with gross contamination of the mediastinum will still require drainage.

The mortality reported so far in spontaneous rupture of the esophagus is 100 per cent excluding the 2 cases mentioned by Graham. Most patients succumb within 24 hours though 1 patient survived 17 days (16). With improved techniques in anesthesia, freer use of blood and plasma for intravenous infusion, and effective chemotherapeutic agents, the limiting factor in the treatment of this condition is that of diagnosis. It does not seem at all unlikely that soon recoveries from this condition will be reported. Prompt resort to the use of roentgenography and esophagoscopy in questionable cases should enable one to establish the diagnosis early. Just as in perforation of the cervical esophagus, prompt operation leads to a low mortality (45) so prompt recognition of spontaneous rupture of the esophagus will be necessary if recoveries are to be obtained.

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DIAGNOSIS AND THERAPEUSIS OF SIMPLE CYSTS OF THE BREAST

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CHRONIC cystic mastitis, a term most generally used for a condition that has been given a confusing nomenclature, has been a subject of controversy for approximately a century. The controversy is not yet settled. The argument is about its relation to cancer; principally between those who believe chronic cystic mastitis is a forerunner of cancer and those who do not. There are those who will not go so far as to commit themselves either way.

There is universal agreement however, even among those who regard chronic cystic mastitis as a precursor of cancer, that the larger cysts are less likely to eventuate in cancer than are the ductal and acinus hyperplasias with small cysts that characterize the histological picture of chronic cystic mastitis. By larger cysts we mean those that are large enough to be discovered by the patient herself or by the examining physician as a lump in the breast.

While the various forms of chronic cystic mastitis are thought to be one and the same thing, the larger clinically demonstrable cysts of simple cystic disease occur more often in breasts of moderate size than in the smaller breasts that are stiff, nodular with saucer-like edge designated as adenosis (Schimmelbusch's disease). In the latter form, in which cysts are more numerous but small, seldom appreciable in size, there is more epithelial activity than in the larger cysts of simple cystic disease.

The early physicians did not consider the larger cysts of simple cystic disease to be dangerous and they treated them by puncture, incision, and occasionally by amputation of the breast.

With the growing use of and greater dependence upon the microscope, the management began to change based upon the interpretation of the findings of an occasional papillomatous process on the walls of a large cyst. This hyperplasia was considered to be at least premalignant, therefore removal of such cysts was advised. This procedure satisfied the earlier pathologist as being adequate treatment even though such breasts in all likelihood contained other cysts of inappreciable size that were left behind and in these cysts hyperplasia was undoubtedly present. In other words that which could not be felt or seen caused no concern. This challenge by the pathologist dominated the surgeon pretty much for 2 generations. In general the surgeon is compelled to be governed in his plan of action by the pathologist's opinion even though his interpretation of tissue submitted may be in error and thus lead the surgeon astray.

It is understandable that there are many instances of cell growth that make troublesome problems of interpretation for the pathologist. To mention an instance, all of us are familiar with the heated controversy among pathologists on the relationship between gastric ulcer and cancer; one extreme claiming that 68 per cent of apparently simple ulcers shows microscopic evidence of cancer, the other extreme maintaining that only 5 per cent of ulcers shows malignant change. Clinical evidence had to decide the controversy. The present generally accepted opinion is that 5 per cent is pretty close to being correct.

In undisputed malignancy lesions the true behavior does not always parallel the histologic picture. As examples, the microscopic picture of a rodent ulcer certainly carries all the earmarks of a rather high degree of malignant growth but we know from experience that it does not metastasize and that its clinical behavior is relatively benign. On the other hand we know from experience that the more innocent appearing squamous cell carcinoma carries a high degree of malignant behavior.

A good example of epithelial overgrowth is seen in Graves' type of toxic goiter a response to stimulation only to regress spontaneously or under treatment toward atrophy and fibrosis. Here again we know from experience that its behavior is not at all that of malignancy. In a similar way the breasts are subjected to strong stimuli (physiologic), during the menstruating years, causing proliferation of cells lining the ducts to form papillae with cells as much as 4 and 5 deep and also proliferation of acinus epithelium which also tend to regress by an involuntary mechanism. These 2 instances then of epithelial overgrowth as they occur in Graves' type of toxic goiter and in

From the Blodgett Memorial Hospital.

the ducts and acini of breasts belong most likely in the same category.

It is noteworthy in reviewing the voluminous literature on the relationship of chronic cystic mastitis to cancer that opinions vary from nil to 20 per cent, which again brings to one's realization a bewildering conflict of opinions. Bloodgood, who had been a contemporary advocate of the casual relationship of chronic mastitis to cancer of the breast, had a change of mind after a more profound study of the subject. He was the first really to challenge, in 2 masterful papers, the concept that had prevailed for many years. In the first in 1921, he reported 128 cases in 3 of which cancer developed; in the second in 1929, he reported 500 cases in only 5 of which cancer developed, an incidence even less than occurs in the absence of cystic disease. His studies are of particular value since they combined and correlated the microscopic picture with the clinical course of the disease in some instances for 29 years.

Bloodgood submitted sections of every type of chronic cystic mastitis to more than 40 consulting pathologists. In the majority of cases there was disagreement. The percentage of those favoring benignancy varied from 20 to 90; furthermore, in tissues and sections that were sent to him for diagnosis he had never received one which proved to be a fully developed cancer that had not been diagnosed cancer, whereas he had received many with the diagnosis of cancer which in his opinion belonged to some form of chronic cystic mastitis, further illustrating the difficulties of the histologic interpretation of the nature of growth—or overgrowth. He concluded by saying that in breast lesions when good pathologists disagree as to malignancy, the patient lives. When there is agreement there is always a large percentage of deaths from cancer.

Reclus, an outstanding student of the subject, in 1883, 38 years before Bloodgood's challenge, also had a change of mind. While he originally believed in the benignancy of cystic disease, he had been influenced by the pathologist to question his stand only to revert to his original opinion after further study of his cases upon learning that those that had been treated conservatively remained well.

In 1934 Campbell summarized the literature and contributed a series of 290 case studies of cystic disease with clinical follow-ups from which he was compelled to conclude that cystic disease is not a precancerous lesion; that malignant changes are no more likely to follow this disease than to occur in breasts that are entirely free from this condition. He includes in this study the adenocystic (Schimmelbusch's disease) variety. Two patients of the 290 cases reported developed breast cancer—0.7 per cent. He contends that the clinical test of time is the true answer to the controversy.

Lewis and Geschickter in 1938 reported 515 cases of cystic disease. Two hundred fifty of this number were followed for more than 5 years. One of this group of 250 died of breast cancer, an incident of 0.4 per cent. From this study it is their conviction that cystic disease is not a precancerous lesion.

Warren in 1940 presented a comprehensive study in which he concluded that 4.5 times as many cases of chronic cystic mastitis subsequently developed cancer of the breast, age for age, as would be expected for the female population as a whole. His studies include all forms of mastitis. He does agree, however, that the large, clinically palpable cysts with atrophy of their lining epithelium are less likely to be followed by cancer than are those with active, often papillary, epithelium and that aspiration of the contents may be a justifiable therapeutic procedure. However, he believes excision to be preferable since it is difficult to determine the exact nature of cell hyperplasia if present.

Thus it is evident that whether hyperplastic cystic disease (large simple cysts, papillary cysts, papillary cystadenomas and duct adenomas) is endowed with malignant qualities or not is a question still not settled. It is however, fair to state that all students of the subject agree that the larger (clinical) cysts are relatively harmless. There are those who believe that they are entirely harmless. Yet in recent times no one seems to have the conviction or courage to treat these large simple cysts by puncture.

For reasons already stated, to remove a large cyst, in which there is little or no hyperplasia of the cells of the walls, while leaving innumerable smaller cysts, also acini and ducts with definite hyperplasia of cells, is inconsistent and unsound reasoning. And to repeat also, that which cannot be felt or seen (still) causes no concern.

That these simple cysts are harmless, in so far as being forerunners of malignancy is concerned, became my conviction 14 years ago. Since that time I have managed simple cysts by puncture as a diagnostic and therapeutic office procedure.

In making the clinical examination the experienced observer can in a goodly number of cases be correct in his conjecture as to the nature of the lump he is dealing with but this conjecture must be verified. A cyst of clinical size if superficially located transmits, as a rule, to the exam-

ing hand, the feel of just what it is—a cyst that is tense but not hard as is a fibroadenoma. While movable, it does not "shuck" around like a fibroadenoma or a lipoma (rare). The deeper lying cyst often imbedded in dense and thickened connective tissue stroma cannot be differentiated by the feel from malignant growths. In either case there is no skin attachment.

The following is the office procedure for diagnosis. In the absence of clinical signs of malignancy the overlying skin is injected with local anesthetic by means of a fine needle; a larger needle is then introduced into the lump. If it is a cyst the plunger of the syringe will be pushed up by the fluid under tension. Pressure with the fingers over the lump area helps to remove the last drop of fluid, so to speak. Few cysts so managed refill. It has been conjectured that it is because of complete emptying that so few refill. From my experience I have learned to form an opinion on the nature of a lump, that is not a cyst. A fibroadenoma is tough like rubber; a thickened wall of a deep seated cyst is likewise rubbery; a medullary carcinoma is soft, as is a lipoma; a scirrhous carcinoma transmits a gritty feel. While this interpretation of "feel" has no special practical value it is a refinement in clinical diagnosis and gives some satisfaction when committing oneself to a definite preoperative diagnosis.

This report of 57 case studies is limited to clinically palpable, large simple cysts exclusive of other forms of cystic mastitis. While the number of cases is not large, its importance lies in the period of observation; namely, 14 years. All have been followed and checked to the present time.

Age incidence. The peak of incidence was in the latter half of the fourth decade. The youngest subject was 27 years of age; the oldest 62 years. In the 26 to 30 year age group there was 1 case; in the 31 to 35 year group, 5 cases; in the 36 to 40 year group, 13 cases; in the 41 to 45 year group, 13 cases; in the 46 to 50 year group, 2 cases; in the 56 to 60 year group, no cases; in the 61 to 65 year group, 1 case.

Child bearing. Twenty-six of the 57 patients had borne children; 30 were childless; in 1 patient, not recorded.

Number of cysts. The 57 patients had a total of 123 discernible cysts; 15 patients had cysts in both breasts. Thirty-three had 1 cyst, 12 had 2 cysts, 2 had 3 cysts, 4 had 4 cysts, 4 had 6 cysts, 1 had 9 cysts, 1 had 11 cysts.

Incidence in right or left breast. The left breast harbored slightly over twice as many as the right; left breast, 29 cases; right breast, 13 cases. In 1 of these patients an early carcinoma was present in the opposite breast. Both breasts were involved in 15 patients.

Location. The upper hemisphere, especially the upper outer quadrant, is more often affected than the lower in the ratio of 3 to 1.

Size. The size of the lumps varied from a hazelnut to a tennis ball; the average from a hazelnut to a walnut. The amount of fluid varied from ½ to 45 cubic centimeters, the average from ½ to 10 cubic centimeters.

Character of contents. Cloudy, grayish with a greenish cast is the usual appearance of the contents; less frequently it has a brownish cast. In 2 cases, the cysts were blood containing; one was excised. The other patient, one of the earlier cases, procrastinated but no trouble followed.

Dry puncture. In 2 cases, deep seated lumps proved on excision to be cysts embedded in dense heavy connective tissue stroma. The needle had gone past the cysts which were small—the size of a hazelnut.

Mastectomy. In 2 cases, multiple large cysts in both breasts embedded in heavy dense stroma, kept on refilling and were painful; these were finally treated by mastectomy. In only 3 other cases, as far as could be ascertained, was there refilling.

Simple cyst and early malignancy. One patient had a cyst in one breast; a small lump in the other that gave a dry tap proved upon excision to be an early carcinoma and was treated accordingly. There was no histologic evidence that the malignant lesion had its origin in mastitis of any form. This was the only malignant growth encountered in the series of 57 cases.

The 57 patients were spared 83 surgical operations in all, since in several cases cysts appeared subsequently.

DISCUSSION

The report of this series of cases may not be sufficiently convincing to settle an old argument. However, study of clinical cases followed through for as long as 14 years, average 7½ years, is strong evidence. The behavior of growth is, in the last analysis, the answer to its nature. It should be more convincing than the evidence of microscopic interpretation of growth which, admittedly, is not infrequently difficult and erroneous.

The nature of every lump in the breast must be determined. (One case of a cyst in one breast; an early carcinoma in the other).

It is said that simple cyst lumps cannot with real certainty be differentiated from cancer by palpation. The answer to that is needle puncture—most simple, an office procedure. It gives identi-

cal information and the same protection to the patient that is obtained by excision. It is equivalent to the discovery by the surgeon that the lump excised is a cyst, whereupon also the surgeon carries the operative procedure no further. In the event of multiple cysts or others to appear, it becomes then a piecemeal, unsatisfying job of surgery. Such instances come to one's attention not infrequently. All in all, in that case, it means the hard and costly way for the patient.

In the 57 cases comprising this study, there was one instance of early malignancy in one breast; a simple cyst in the opposite one, in a patient 50 years of age—an incidence of 1.7 per cent; normal incidence said to be approximately 2 per cent.

The fact that whereas chronic cystic mastitis is one of the commonest lesions found in the female breast, yet cancer of the breast occurs in relatively only a small percentage (approximately 2 per cent) of individuals, is in itself strong evidence to absolve chronic cystic mastitis from being a forerunner of breast carcinoma.

The more recent exponents of the benignancy of simple cysts of the breasts notably Bloodgood, Campbell, Lewis and Geschickter still advocate excision for the most part. The author's experience is in agreement as to the benignancy of simple cysts but he differs only in the management of them, in that he has practiced simple puncture consistently for the past 14 years as a diagnostic and therapeutic office procedure. The patient is thereby afforded protection equal to that of excision. At the same time it has spared her the ordeal of an operation—or even operations, with all it entails—not excluding hospital cost and surgeon's fees.

NOTE.—Anyone not trained, skilled, and experienced to do radical breast surgery should do neither a simple puncture nor a biopsy.

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SURGICAL ASPECTS OF TRENCH FOOT

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ON February 7, 1945, this former Army Service Forces Convalescent Hospital was designated a general hospital, largely for the treatment of trench foot and frostbite injuries. In addition, a portion of the camp facility has been rebuilt to function separately as a convalescent hospital, and the two institutions, grouped together, comprise a hospital center for the study, treatment, rehabilitation, and disposition of trench foot cases. During the 4 months between February 7, 1945, and June 7, 1945, 4,892 soldiers with frostbite and trench foot were admitted to this hospital center. Many were later cases on arrival; more, however, were ambulant. Originally, all were assigned to the medical service. There, the great majority became not only ambulant, but reached a maximum degree of hospital benefit; 264 had varying degrees of persistent gangrene, and it was a policy of this medical department to transfer these cases to surgery after they had attained maximum improvement on the medical regimen. The time involved in gaining this improvement varied from 4 weeks to 2 months.

At this point, the current concept of trench foot might be briefly mentioned because it is upon it that the rationale of treatment is based. Main factors concerned in the development of this injury apparently are the following: (a) Exposure to cold but not necessarily freezing weather for (b) varying lengths of time from a few hours to, usually, several days, and occasionally much longer periods, during which (c) the feet are wet or at least damp, and, in addition, are (d) constricted by foot gear that is not too tight for ordinary wear but is too tight for feet beginning to swell, and finally, but not the least factor in importance, (e) prolonged relative immobilization of the whole body, particularly the dependent legs and feet. This prolonged exposure, in which tight shoes and wet socks enclose cold and immobile feet, brings about trench foot. Perhaps, in some cases, all these factors need not be present to bring it on, but if all are present, its development is quite likely. Relative malnutrition and fatigue may occasionally be additional factors.

Early, it is known, there occurs reflex capillary and arteriolar vasoconstriction. Anoxemia of all tissues of the toes and foot follows for this reason, and also because of lessened oxyhemoglobin dissociation which occurs at low temperatures. Anoxemia leads to damage to all affected tissues. At this stage, the feet are insensitive but hours later, on removing the feet from the faulty environment, a contrasting reaction occurs. Feet and toes become red and hot to palpation, and burning hot subjectively. They swell and the toes become dusky. Hemorrhagic blebs may appear on both soles and dorsum of feet and toes. At this early stage it is believed the capillaries are dilated, their endothelium damaged and the capillary stream slowed. Increased capillary permeability leads to increased exudation into interstitial and intercellular spaces of the foot. So much fluid escapes that the edema may appear in the epidermis as blebs. The anoxemia, already aggravated by edema, is further increased by thrombosis of many small arterioles, consequent upon their damaged endothelium and vascular stasis. If during this stage the foot be heated or exercised as a therapeutic measure, all these unfavorable factors become aggravated. Local nerve tissue also suffers from anoxemia. Due either to irritation or destruction of peripheral nerve elements, vasomotor changes follow, also sensory and, presumably, trophic changes as well. All other structures also suffer. As the edema subsides, an inflammatory type of repair occurs in the viable areas above the demarcation line. The end-result is fibrosis, not only of the subcutaneous tissue where fat has been lost, but of intrinsic foot musculature as well. Tendon sheaths become involved in adhesions. Joint capsules acquire adhesions and frequently there results a chronic noninfectious arthritis and synovitis. Periosteum may thicken and bone cortex becomes decalcified. Marrow may become replaced by fibrous tissue. Toe nails exhibit evidence of an arrest in growth. A transverse raised ridge may later be seen growing out of the nail bed and for many months the nail is thickened and deformed. Typically a mixed type infection exists at the demarcation

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The following additional officers participated in the surgical care of these cases and contributed to the opinions and evolution of surgical principles outlined in this paper: V. L. Hart, Lieutenant Colonel, M.C., Seventh Service Command orthopedic consultant; Harry W. Woodward, Lieutenant Colonel, M.C.; Harold L. Gordon, Major, M.C.; Jesse F. Harrold, Major, M.C.; Norbert J. Kulzer, Captain, M.C.; Alvin R. Megahan, Major, M.C.; Gordon S. Owen, Captain, M.C.

Fig. 1. Photograph of gangrene of finger tips resulting from frostbite.

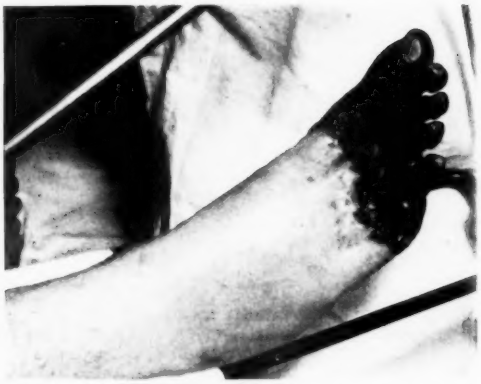

Fig. 2. Trench foot gangrene of distal portion of foot associated with compound comminuted fracture of femur.

line, but this does not often spread far into nearby viable tissues. The distal part remains black, dry and leathery. Less severe cases have only gangrene of the skin, the deeper structures remaining viable. Such toes cast off their dead shell after new skin has regenerated beneath it.

In contrast to these severe cases, the greater number do not eventuate in gangrene at all. Although most feet become anatomically normal after several weeks or months, many continue painful on weight bearing and sensitive to cold for an indefinite period. This report is not concerned with these lesser degrees of trench foot, which were not under our direct observation.

Most surgical cases had only distal parts of some toes devitalized and consequently required partial toe amputations. A small number lost all toes on at least one foot, and some lost one or more metatarsal heads as well (Table I). In a few cases, and these were usually severe ones (Figs. 1 and 2), trench foot was associated with leg wounds, such as a compound fracture or trauma to the femoral artery. In these circumstances, the soldiers had been lying on cold ground, from several hours to as long as 3 days.

Preoperative routine. The patients were given every chance to improve on conservative management. Active and passive exercises were given to the toes, many of which were atrophic and fixed in partial flexion. The patients were instructed as a group forcefully to flex and extend their toes—using their fingers to do it. This ward exercise, done several times daily, was combined with a lanolin lubricated massage to relieve the dry scaling frequently seen on these toes. Adequate passive and active motions tended to free adhesions about tendon sheathes and joint capsules, and many toes that would otherwise have had to be amputated before shoes could be worn, were thereby saved. In addition to exercises to loosen up fibrotic toes, active foot and ankle exercises were employed to prevent contracture of the tendo achillis and gastrocnemius soleus muscle group with consequent talipes equinus and pes cavus. Other frequently employed types of physical therapy, and whirlpool baths, in particular, have proved of no use in most cases.

The usual preoperative laboratory work included smears and cultures taken at the demarcation line. Not only was the bacteriological flora identified but, if applicable, penicillin sensitivity tests were reported at the same time. This entailed only an additional day's delay in receiving the report. Of 95 cases in which penicillin sensitivity composed part of the bacteriological study

TABLE I.—DISTRIBUTION OF GANGRENE

	Cases
Partial gangrene of toes	197
Loss of all toes of a foot at proximal joint	11
Loss of one or more metatarsal heads	32
Loss of all metatarsal heads	35
Loss requiring transfer to center for "below knee" amputation	2
Gangrene with loss of heel plantar skin	0
Gangrene of fingers (Fig. 1)	3

TABLE II.—BACTERIOLOGICAL FLORA

Resistant to 1 unit penicillin per c.c.:	Case
Hemolytic Staphylococcus aureus	2
Bacillus coli	
Susceptible to 1 unit penicillin per c.c.:	
Nonhemolytic cornebacterium	1
Hemolytic diphtheroid cornebacterium	
Beta hemolytic streptococcus	1

Fig. 3. Left, This great toe had formerly been black up to the metatarsophalangeal joint. The gangrene was only superficial and was cast off like a glove. (Note the transverse ridge on the nail.)

Fig. 4. Thin grafted skin is unsatisfactory permanent covering for end stumps. It ulcerates when weight bearing is attempted.

it was found that most wounds harbored at least 2, and often 3 or 4 organisms. Table II shows the spread of the flora.

The trench foot cases were routinely given a preoperative injection of 50,000 units of penicillin intramuscularly when they received preoperative medication. The routine use of sulfa drugs was not encouraged because of the needlessly increased risk of reactions associated with their use.

Roentgenograms of the involved foot were requested as part of the preoperative work.

Surgical principles. Since no clear cut precedent had established all the surgical principles concerned in the surgical treatment of trench foot, the general plan of treatment underwent evolution as experience accumulated. For instance, it was deemed wise to delay surgery until all possible spontaneous recovery had occurred. Many soldiers with toes black on admission here, progressed so favorably on conservative care, that within 2 to 4 months they lost the gangrenous tissue (Fig. 3). A considerable number of these soldiers now have their toes and feet intact. Had they been operated upon early, they would have suffered an unnecessary loss which, originally, could not have been well prognosticated. A few soldiers have been admitted here who were subjected to early amputation elsewhere, and they usually seemed to present greater loss than one would expect should have occurred.

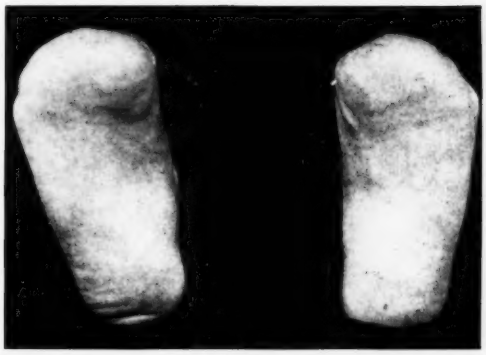

Fig. 5. Above, A flap of normal weight bearing plantar skin and subcutaneous tissue is required as cover for good end stumps.

Fig. 6. All metatarsal heads have been sacrificed to provide good end stumps from plantar skin. This soldier runs and walks surprisingly well.

Another surgical principle that emerged with experience, was concerned with the inadvisability of depending upon grafted skin to cover stumps of feet. The advantages of retaining all possible foot length are obvious, but the attempt to accomplish this by grafting skin to an exposed stump results in a poor foot. Such a foot with an end stump covered with thin skin has no subcutaneous tissue to protect the superficially palpable bone ends (Fig. 4). Weight bearing

Fig. 7. Skin traction apparatus to assist in closing skin flaps and in preventing skin retraction.

stretches this unpadded skin, which is of low vitality, and it is prone to break down and become the site of an ulcer. Several such patients received here were submitted to corrective surgery. Where good plantar skin flaps, having adequate subcutaneous tissue, were brought up to cover end stumps, good functional feet were obtained. This plantar type of skin and subcutaneous tissue is highly specialized for weight bearing and when pads of it cover bone ends, the stumps are well protected against trauma.

In order to gain this type of end covering pad when gangrene has extended proximal to the webs of toes, it was sometimes necessary to foreshorten metatarsals by removing their heads. Contrary to general surgical opinion, removal of the first metatarsal head, or the fifth—or any—or all five, does not usually too seriously handicap function of the foot. As a surgical principle these heads were removed without hesitation, if that was necessary to obtain enough skin and subcutaneous tissue, particularly from plantar flaps, to cover stump ends (Figs. 5 and 6).

Another surgical principle concerned the method of obtaining closure of the stump. All of these feet were infected, in the sense that bacteriological smears and cultures could be obtained from the demarcation line, but few were chronically inflamed in the area of proximate viable tissue. At first, true guillotine type amputations were done. These required secondary surgery when prolonged skin traction was not completely successful, and the resultant secondary surgery meant additional loss of toe or foot length. Consequently, it became general practice, even o toes, to leave generous flaps to cover the stumps Where possible, these flaps were fashioned fro the plantar surface. Secondary closure, at firs done on the fifth day if the stump was clinically free from infection, was advanced with increasing experience, to the fourth and third days, unti ultimately, primary closures were done. This wa safe because of the postoperative routine pres ently to be described.

In some early instances when the first metatarsal head was removed, the sesamoids were allowed to remain. They became secondarily chronically infected and were the source of draining plantar sinuses. Even in some disarticulations in which the metatarsal heads were not removed, this was still true. Because these sesamoids had to be removed later, it became a policy to remove them carefully at the original operation, except in those cases in which the metatarsal head was allowed to remain, and in which, further, the sesamoids were safely deep from the edge of the plantar skin flap and clearly surrounded by viable tissue. Under these latter circumstances, they were not disturbed, and none so selected gave further trouble. In fact, they no doubt were useful in helping make up the normal bulk of the resilient pad beneath the first metatarsal head.

An additional surgical principle that frequently proved helpful in taking strain off a suture line and preventing retraction of skin, or occasionally in aiding the approximation of a secondarily infected stump, was skin traction (Fig. 7). Traction was provided by gluing a short length of stockinet on to the foot with ace adherent. Counter traction was obtained by suspending a spring metal hook over the stump and anchoring this hook into a plaster boot. The boot extended to just below the knee, and held the foot at a right angle. This boot loosely covered much of the stockinet but was quite free from fixed contact with it. A spreader with eight to ten rake teeth was hooked into the stockinet and suspended from the boot hook by means of a short length of rubber tubing under considerable tension.

In a few instances having short foot stumps, n additional plantar skin was available to bring u over stump ends. Some of these feet with goo ankle motion and adequate plantar walking su face were not condemned to amputation. Instead full thickness skin flaps were employed to cove the stumps. Either cross leg flaps or jumped tube were used. Similar plastic procedures were re quired to cover a few denuded heels.

Additional operative procedures, usually though of in association with trench foot, have not bee

done here. There has been no place for either sympathetic block or sympathectomy.

Surgical technique. All amputations were done under pentothal-oxygen anesthesia. Incisions were made just proximal to the typically narrow zone of reaction on the plantar skin. A longer plantar flap and shorter dorsal flap made possible a dorsally placed scar line. Because of lessened tissue viability, together with the bacteriologically positive conditions present, it was a principle not to place buried sutures, absorbable or otherwise, within the amputation stump. As a matter of fact, although bleeding was usually brisk, it was practically never necessary to ligate a vessel.

The periosteum was left undisturbed prior to bone resection. The additional trauma to surrounding soft tissues involved in freeing this periosteum did not aid in the kindly healing of tissues which were, at best, handicapped in viability. Perhaps worth recording is the impression that these stumps and flaps were sometimes not free in their bleeding; that frequently the cancellous bone, on being sectioned, stayed white and free from oozing; that the cortex was usually extremely soft, but at other times was normally dense and brittle and, that now and then, the digital arteries were grossly thrombosed. Regardless of these not so favorable findings, the wounds healed kindly, if somewhat slowly. Closure was either primary, or delayed 2 or 3 days, depending on the gross appearance of the skin flaps. Sutures were fine black silk, sparingly used.

Postoperative routine. Following operation, saline dressings were used when a delayed primary closure was being done, and, simple dry dressings for primary closures. Sutures were allowed to remain up to 6 days in the presence of mild postoperative redness and up to 10 or occasionally 12 days if the skin was entirely free from reaction. Sometimes, when it became necessary to remove sutures before healing was complete, adhesive butterflies were useful, but, if left on too long, maceration and undue pressure effects were found beneath the tape. For large stumps in these circumstances, the traction cast previously described was useful.

Postoperatively, penicillin was given in 25,000 unit doses intramuscularly every 3 hours for at least 300,000 units and frequently for 5 or 6 days, or until the sutures had been removed.

The patients were kept off their feet quite a bit longer than one might think necessary. Even after sutures had been removed and the toes or foot stumps seemed well healed, a soldier frequently stubbed a toe, and this sometimes developed a reactive hyperemia or even an acute cellulitis which was slow to resolve. It seemed the tissues lacked resistance to trauma, and being traumatized, lacked resistance to infection. For this reason, a policy of prolonged observation was maintained after definitive treatment had been completed. During this time, of course, the same group physical therapy employed preoperatively, was resumed in normally convalescing cases. Each patient was required to complete 30 days without incident, either on convalescent furlough at home, or at the convalescent hospital, before being given a Certificate of Disability Discharge.

Fig. 8. Prosthesis built on insole for use in standard type shoe.

A simple prosthesis has been evolved for all patients who have lost three or more adjacent toes, and for all patients who have lost the metatarsal heads (Fig. 8). Basically, it is a shoe insole, often combined with a longitudinal arch support, and having built on to it a felt block to fill in the space between the foot stump and shoe tip. That part of the felt block nearest the stump is faced with lamb's wool. The rest of the block is covered with thin soft kid leather. In those patients not requiring arch supports, insoles are of thin leather. To prevent the shoes from flexing too much on weight bearing, the tips of the soles are opened and a flat spring steel insert is slipped lengthwise into the soles. By means of these prostheses, the owner can purchase standard shoes, and can wear them without complicated alterations. Using these prostheses, wearers walk more comfortably and more naturally than they do without them.

Complications. In about 20 per cent of the cases a slight local redness appeared postoperatively. This transient cellulitis was usually not severe enough to require the removal of sutures,

and penicillin usually seemed to bring it quickly under control. When it was inadequate, one of the sulfa drugs sometimes had to be substituted. The cellulitis then promptly disappeared.

Not to be confused with this mild postoperative cellulitis was a different phenomenon of rather similar appearance. When these soldiers began to regain use of their feet, the tissues about the stump ends not infrequently became quite red and angry looking. This condition was associated with neither pain, heat, or swelling, and on resting for a few hours, it completely disappeared. This reactive hyperemia, in response to trauma, as brought about at first by weight bearing, tended to lessen and finally disappear as the days went by. It proved to be of no serious import, but when present, walking was somewhat curtailed.

Except for the mild local cellulitis previously mentioned, postoperative difficulties were few. There were 23 instances of infection with demonstrable pus. These included 4 cases of acute lymphangitis and 1 lumbrical space abscess. Chronic osteomyelitis was surprisingly rare. There were only 5 cases, 1 of these being associated with trench foot in a compounded injury.

Varying types of dermatitis were troublesome in 9 cases. Among these, a temporary generalized urticaria due to penicillin occurred twice, a localized staphylococcus pyoderma was present twice, and a chronic eczematoid dermatitis once. This latter involved all the toes, and the skin was chronically reddened, dry, and scaly.

In these surgical cases, sweating was no problem. In trench foot reports by some, much emphasis has been placed on cold sweating feet. When it is recalled that this phenomenon is part of the characteristic picture of "battle fatigue" and that sweating is frequently present in soldiers who do not have trench foot, and further, that many soldiers with trench foot also have "battle fatigue," one can hardly specify sweating feet as exclusively part of the trench foot picture. In these cases of most severe trench foot, if gangrene is any criterion, there was no more sweating than would be found among a like number of soldiers convalescing from other types of injuries.

Psychologically, the loss of some or all toes, or even the distal portions of one or both feet, has presented no problems that have not been satisfactorily mastered. Probably this is because the soldiers are treated in wards where they are surrounded by others with surgical trench foot.

Disposition. Final disposition has been made on most of these cases. None were returned to duty whose feet were not anatomically and symptomatically normal. Therefore, all here reported either have been given certificate of disability discharges, or will be given them when they have reached maximum benefit of hospitalization. Before being discharged, information regarding future care of the feet is given in group talks. All are told that they will continue to improve gradually for as long as 2 years, but that at first they must take measures to guard their feet from undue trauma.

SUMMARY

From a total of 4,892 soldiers with trench foot admitted, an experience with 264 cases requiring surgery is recorded. Many additional patients had only a temporary superficial gangrene of the skin of the toes, which spontaneously recovered.

Most patients lost only parts of several toes. Eleven, however, lost all toes of at least one foot, and 32 lost some or all the metatarsal heads as well. With increasing experience, it was found useful to adhere to certain surgical principles. Surgery was delayed until all possible spontaneous recovery had occurred. This saved toes and feet. Grafted skin was found unsuitable for covering stumps. The advantages of covering a stump with a flap of normal weight bearing type plantar skin outweighed the advantages of retaining metatarsal heads. Contrary to general surgical opinion, removal of some or all metatarsal heads was found not too seriously to handicap the function of the foot. At operation, the disadvantages of tying bleeders and of employing a periosteal elevator are mentioned. Sesamoids were removed only when infected or when it was expected they would become infected and cause a postoperative draining sinus. When penicillin was routinely administered, both preoperatively and postoperatively, it was found practicable primarily to close the skin flaps fashioned at the time the gangrenous toes were removed. Infection was rare, and there was no correlation between the frequency of infection and the types of infecting organisms. An effective method of skin traction was sometimes used to help take the strain off suture lines and prevent skin retraction. A postoperative reactive hyperemia on a traumatic basis is described and contrasted to postoperative cellulitis due to infection. Postoperative complications were relatively infrequent. Preoperative and postoperative active exercises helped to prevent shortening of heel cords and correct fibrotic contracture of toes. A prosthesis designed to be part of a shoe insert, often combined with a longitudinal arch support, is described.

THIOURACIL WILL NOT REPLACE THYROIDECTOMY

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AFTER 2 years of clinical experience with thiouracil in the treatment of hyperthyroidism, it is my belief that this drug will not replace thyroidectomy, despite the enthusiastic reports accorded it in the literature. Such claims that thiouracil is the method of choice in the treatment of toxic goiter are unscientific. The facts were better stated in an editorial in the *Journal of the American Medical Association*, September 16, 1944, as follows: "Whether or not thiouracil will prove to be a satisfactory substitute for surgical treatment of toxic goiter cannot be stated on the basis of present limited experience. The drug promises to be of great value in cases in which operation is inadvisable or contraindicated."

Thiouracil will never replace thyroidectomy in the large group of cases commonly designated as multiple toxic adenomas or adenomas with hyperthyroidism. Whether one chooses to designate this group of cases as a distinct clinical entity after the method of Plummer or whether they be considered together with exophthalmic goiter as merely a variation of the same disease makes little material difference. Adenomas are essentially neoplasms and are prone to affect the patient in many other ways than through their toxicity and, as such, require surgical removal. One should surely hesitate to advise the relief of hyperthyroidism in a case of this kind and then see the patient later succumb to a malignancy or the effects of an intrathoracic goiter. Consequently, fully 40 per cent of the so called toxic goiters have been eliminated from the category of successful treatment by thiouracil.

There remains the group of cases commonly spoken of as exophthalmic goiter (diffuse toxic goiter—Basedow's disease, Graves' disease, etc.). In this type much more investigation and study will be required before any considerable number of cases may be judged as cured. In a small series of 35 cases I would hesitate to say that any might be considered as cured judging from the short period of time they have been under observation. Furthermore, experience seems to favor the opinion that the majority of cases of this type should not be subjected to treatment with thiouracil on the basis of our present knowledge of the drug. In a recent discussion of the subject mention was made of the fact that 27 fatal cases of agranulocytosis occurring from the use of thiouracil had been reported up to that time. This is an exceedingly high mortality in comparison with that for the operative treatment of the average case of exophthalmic goiter in the hands of the experienced thyroid surgeon. Patients residing at a distance find it inconvenient to report for frequent blood counts, and thus is augmented the possibility of the development of agranulocytosis.

If the health of the average patient suffering from exophthalmic goiter may be restored by surgery within a few weeks and he resume his usual duties with a normal metabolism, there seems little justification in subjecting him to months or years of therapy with an admittedly dangerous drug. Furthermore, we agree with Lahey[1], who is of the opinion that since thiouracil in no way changes the histologic picture of exophthalmic goiter, it is not a drug to be employed as a curative agent.

There are, however, certain indications in which the risk of using thiouracil seems definitely justified in comparison with the risk of surgery. These indications previously indicated[2] might be enumerated as follows:

1. Advanced cases of multiple toxic adenoma in which there is no response to iodine.
2. Cases of toxic adenoma complicated by such conditions as myocarditis, fibrillation, hypertension, diabetes, and decompensation.
3. Cases of severe exophthalmic goiter in such patients as the very young, the aged, the debilitated, and the decompensated.
4. Cases of exophthalmic goiter in whom because of pregnancy, previous nerve injury, infections, or other diseases it may be desirable to delay surgery and yet not continue on iodine.
5. Cases of iodine-fast exophthalmic goiter.
6. Cases of recurrent or persistent hyperthyroidism.

Our experience in all these types of cases with thiouracil has proved encouraging. Probably the gravest risk in the surgical treatment of hyperthyroidism has been brought under control, namely, the advanced cases of toxic adenoma in which a two stage thyroidectomy has usually

The Freda Meyers Nishan Foundation for the Study of Goiter of the Jackson Clinic.

[1]Lahey, F. H. Kan. City M. J., 1945, July-Aug., p. 11.
[2]Jackson, A. S. Am. J. Surg., 1945, 67: 467-478.

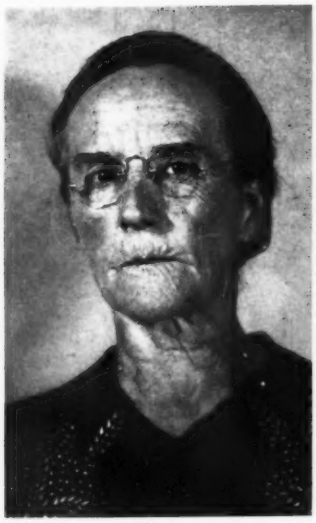

Fig. 1.

Fig. 2.

Fig. 3.

Fig. 1. No. 124-539. Case of multiple, toxic adenoma of the thyroid. Basal metabolic rate, +60 per cent. Primary thyroidectomy following thiouracil preparation. Patient did remarkably well and had no postoperative reaction.

Fig. 2. No. 117-399. Case of advanced, multiple, toxic adenoma of the thyroid with chronic myocarditis. Poor surgical risk. Right lobectomy 1943, but too bad a risk for second stage. Symptomatically controlled with thiouracil. Basal metabolic rate, 0.

Fig. 3. No. 124-564. Case of exophthalmic goiter in an 11 year old child. Clinically, not satisfactorily controlled with thiouracil. Two stage thyroidectomy. Preoperative basal metabolic rate, +38 per cent, pulse 111, weight 73 pounds. Six months later basal metabolic rate, −1 per cent, pulse 80, weight 85 pounds.

been required to safeguard the patient. After several weeks of preparation with thiouracil, I have successfully performed a one stage thyroidectomy in 12 of these cases. Likewise, in several cases in which the systolic blood pressure was in excess of 225 and in which the threshold of cardiac reserve was small, thiouracil has appeared to reduce the risk considerably.

There has been an opportunity to use the drug in children in only 3 patients having exophthalmic goiter. An 11 year old child was on treatment for 13 months and now shows an unfavorable response since the drug was withdrawn 3 months ago. Another child of a similar age was kept on the drug for several months with unsatisfactory results and then underwent a successful two stage thyroidectomy. A third child is now on treatment with insufficient time elapsed to draw any conclusions.

An interesting case is that of an 18 year old girl who sustained a bilateral adductor laryngeal paralysis, the result of a thyroidectomy performed previous to her admittance to the Jackson Clinic on April 1, 1943. In December, 1943, treatment with thiouracil was started because she had had a recurrence of the hyperthyroidism and because the risk of surgery was out of proportion to the danger of using the drug. Three months ago (July, 1945) therapy was discontinued, and so far the patient has retained her weight and a normal metabolism.

At the present time I have under observation a woman who is 8 months pregnant and who has a moderately severe exophthalmic goiter. In the past I have performed thyroidectomy in a series of 15 cases of this type with no mortalities in either the mother or child. However, in none of these cases was the patient so near to term, the average being 5 months. Consequently, it seemed inadvisable to carry these patients along on iodine until the time of delivery because of the danger of inducing an iodine-fast state. The case now under treatment has been on thiouracil for 2 months and is in excellent condition. Probably thyroidectomy will be performed about a month after delivery.

The term "iodine-fast" exophthalmic goiter was suggested by the writer at the 1931 meeting at Denver of the American Association for the Study of Goiter.[1] At the time it was described a

[1]Jackson, A. S. West. J. Surg., 1932, 40: 142-149.

Fig. 4.

Fig. 5.

Fig. 6.

Fig. 4. No. 113-573. Case of recurrent exophthalmic goiter with bilateral adductor paralysis of the recurrent laryngeal nerve. Thyroidectomy performed elsewhere. Symptomatically controlled on thiouracil since December, 1943.

Fig. 5. No. 123-459. Case of exophthalmic goiter. Basal metabolic rate, +41 per cent, pulse 124. Excellent preoperative response to thiouracil and Lugol's solution. One stage thyroidectomy; very little reaction. Six months after operation, basal metabolic rate, +11 per cent, 20 pound weight gain.

Fig. 6. No. 124-717. Case of multiple, toxic adenoma thyroid with diabetes. Prepared with thiouracil. Primary thyroidectomy.

a new syndrome, one might even say a distinct clinical entity, in which all the classical signs and symptoms of exophthalmic goiter may be absent. Cases are seen in which there is no thrill, no bruit, no tremor, no exophthalmos, and no weight loss. In fact, these patients may even gain in weight. The gland, instead of being firm, smooth, and symmetrical, is nodular and irregular from prolonged, partially controlled hyperthyroidism. There is no clinical response to iodine. Surgeons have come to look upon these cases with considerable apprehension and to consider the surgical risk as comparable to that in advanced cases of multiple toxic adenoma. Usually a two stage thyroidectomy has been required and even then the reaction that followed a lobectomy was often as severe as in a similar operation performed in the era previous to the use of Lugol's solution.

Thiouracil has proved a boon to the patient and surgeon alike in the treatment of these patients. In our clinic 5 patients with this type of thyroid disease have been operated upon—a primary thyroidectomy being done after several weeks of preparation with thiouracil. All of them showed no more than the average reaction to operation.

Those working in the field of thyroid surgery have never felt that thyroidectomy was the ideal method of treatment of exophthalmic goiter. One speaks of a cure in this disease just as he does in the treatment of cancer, tuberculosis, or syphilis because of the well known tendency in some cases of the disease to recur months or years after an apparently successful thyroidectomy. It has been the hope of all who have studied the problem of hyperthyroidism that some less radical and more satisfactory method of cure might be discovered. When Plummer, in 1922, brought out the use of Lugol's solution, it was thought by some that this might effect a cure. In a few cases of recurrent or persistent hyperthyroidism this has proved to be true, but the danger of producing an iodine-fast state in the primary case has eliminated its use for this purpose. Despite this often reiterated warning by thyroid surgeons, many such cases are still confronting the surgeon.

Crile advocated denervation of the adrenal glands as a method of cure, and at first this operation was enthusiastically received only to fall into disrepute as the results did not justify the procedure.

Now thiouracil is brought forth as a method of replacing thyroidectomy in the treatment of hyperthyroidism. Chemotherapy may prove to be the solution to the successful treatment of hyperthyroidism, cancer, and many other diseases, but as yet it has not attained the success of the sulfonamides in the infectious conditions. It now appears that thiouracil has been under sufficient clinical investigation to state that it will supplement but not supplant thyroidectomy in the treatment of hyperthyroidism. That it may effect a cure in incipient and milder cases of exophthalmic goiter is still to be proved. It has already proved a most valuable adjunct to surgery in the treatment of hyperthyroidism. There is every hope that thiouracil will give way to some less toxic but still more efficacious drug in the treatment of hyperthyroidism. Its discovery is a step forward, and those dealing in the problems of hyperthyroidism are greatly appreciative of investigators who have made thiouracil available.

CONCLUSIONS

Two years of study of the use of thiouracil in the treatment of hyperthyroidism have shown that it will not replace thyroidectomy.

Thiouracil will supplement but not supplant surgery in the treatment of toxic goiter.

It has been noted that 27 deaths have already been reported as occurring following the use of the drug.

Thiouracil cannot effect a cure in toxic adenoma.

Six types of cases are listed in which thiouracil has proved of definite value.

Thiouracil may prove of curative value in the treatment of exophthalmic goiter, but sufficient time has not yet elapsed to warrant such a conclusion.

While the use of thiouracil has proved an important forward step in the treatment of hyperthyroidism by chemotherapy, it is desirable that a less toxic drug be discovered.

FEEDING TUBE STENOSIS OF THE LARYNX

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THE indwelling nasal tube, employed chiefly for gastric decompression or for feeding purposes, is so widely used that it is important to draw attention to the possibility of laryngeal damage it may produce. Instances of this complication are relatively uncommon, but, when they do occur, constitute a difficult therapeutic problem. The indwelling tube in the cases to be discussed was the cause of erosion of the anterior esophageal wall at the level of the cricoid cartilage, with subsequent inflammation of the laryngeal cartilages and their ultimate necrosis. This pathological sequence led to a collapse of the airway and a severe, chronic laryngeal stenosis which required tracheotomy and months or years of subsequent treatment. This discussion is presented to call attention to this serious complication and to stress the predisposing as well as exciting etiological factors responsible for it. Through an understanding of these factors, the recognition of early symptoms and the prompt execution of simple corrective measures will prevent the development of this type of chronic laryngeal stenosis.

PRESENTATION OF CASES

CASE 1. N.G., a 52 year old white Polish housewife, was seen first at the Research and Educational Hospital in March, 1939. She was found to have diabetes mellitus and a large, cystic, firm thyroid without toxic symptoms, but with definite compression symptoms. Her basal metabolic rate was +11 and her fasting blood sugar was 333. She was placed under medical management until August, 1939, when a subtotal thyroidectomy was performed. She had no hoarseness or breathing difficulties following this procedure, and her recovery, while slow, was uneventful. In April, 1940 the patient developed severe pain in the right lumbar area which radiated anteriorly to the right lower quadrant and into the vulvar area. The pain was severe, constant, sharp, and was associated with nausea and vomiting. The pain apparently was an aggravation of a dull pain of which the patient had been complaining for 4 months. On admission her temperature was 101.6 degrees; white blood count, 19,300; and her blood sugar was 250 milligrams per cent.

A tentative diagnosis of an appendiceal abscess was made, and an indwelling duodenal tube was introduced April 17, 1940. The patient was very intolerant of the tube because of discomfort and pain, and made frequent attempts to remove it manually or by vomiting. It was left in place for 4 days. No abdominal surgery was performed as the diabetes was out of control although she received adequate insulin and intravenous glucose therapy. By the time the tube was removed the diabetes had been controlled and the abdominal pain had almost completely subsided. However, the patient complained of an increasingly severe sore throat which began at the time the tube was introduced and continued even after it was removed. She would take only liquids, refusing solids because of pain on swallowing. Hoarseness developed after the tube was removed. The tenderness in the right lower quadrant gradually decreased, and her problem became one of respiratory difficulty. An indirect laryngoscopy on May 4, 1940, revealed considerable edema of the vocal cords; it was difficult for her to make audible sounds.

By the sixth week of hospitalization, pain in the right side of the neck and respiratory difficulty had increased markedly. The larynx was more edematous, especially posteriorly; the arytenoids were large and boggy with edema, and the motility of the cords was impaired by the edema, but no paralysis was present (Fig. 1). A tracheotomy had to be done June 3, 1940. Clinical improvement was slow and x-ray films of the neck demonstrated a progressive destruction of laryngeal cartilages as well as a spread of the laryngeal inflammatory process to the soft tissues of the retroesophageal space (Figs. 2 and 3). Chemotherapy was employed, but because of the patient's generally poor condition and the difficulty in controlling her diabetes, extensive surgery of the larynx seemed contraindicated at this time. Purulent drainage into the larynx persisted for 2 months before gradually subsiding. Subsequent treatment has consisted of two series of laryngeal dilatations under direct laryngoscopy. At present the patient still wears her tracheotomy tube, but can keep it corked for several hours at a time (Fig. 4). Her voice is husky but audible.

Thus, this patient, a diabetic, had a feeding tube in place for 4 days during which time it caused discomfort, sore throat and pain, and vomiting had occurred. Laryngeal edema subsequently developed, and a tracheotomy became necessary 7 weeks following removal of the tube. The patient now has a chronic laryngeal stenosis which is responding slowly to treatment.

CASE 2. Mrs. E. B., white, 48 year old housewife. In September, 1940 this patient had a gastroenterostomy and a stormy convalescence followed. A nasal tube was in place 17 days. During part of the time the tube was in place the patient complained of a severe sore throat and pain extending into her ears. This subsided temporarily and the patient was discharged from the hospital. The pain recurred in 2 weeks and became very severe; it was followed by laryngeal edema which increased gradually. Four weeks after leaving the hospital readmission became necessary because of respiratory distress, and an emergency tracheotomy had to be done 24 hours later. Improvement was gradual, and the patient was extubated 5 months later. During the ensuing 3 years dyspnea with exertion was

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Fig. 1. Case 1. Lateral roentgenogram of the neck May 28, 1940. A feeding tube had been in place 4 days, 6 weeks prior to this date. Significant postcricoid and arytenoid edema are visualized.

Fig. 2. Case 1. Lateral roentgenogram of the neck June 10, 1940. The cricoid and thyroid cartilages show some mottling and the postcricoid thickening has increased. The airway subglottically narrowed; patient tracheotomized.

noted, progressing gradually to the point of severe respiratory embarrassment with the least exertion. She was admitted to St. Luke's Hospital at this time, and both the mirror and roentgen examinations demonstrated an extreme subglottic stenosis (Fig. 5). The motility of the larynx was impaired because of cicatricial contractures, but the configuration of the vocal cords was essentially normal. A second tracheotomy was done in March, 1944, and the larynx dilated repeatedly by direct laryngoscopy. The tube was removed in September, 1944, after the patient's

Fig. 3. Case 1. Lateral roentgenogram of the neck July 5, 1940, showing partial destruction of the cricoid cartilage and obliteration of the subglottic airway.

Fig. 4. Case 1. Lateral roentgenogram of the neck afte subsidence of the acute inflammatory process. Laryngea dilatations have resulted in improvement of the airway

Fig. 5. Case 2. Lateral view of the neck showing partial destruction of the cricoid cartilage, postcricoid thickening, and reduction in the airway at the level of the cricoid cartilage. Arrow indicates the stenosis at level of cricoid.

Fig. 6. Case 3. Lateral view of the neck showing the postcricoid thickening and narrowing of the airway at the level of the cricoid cartilage. The principal area of stenosis was immediately subglottic.

airway was tested by plugging the tube night and day for several days.

In this case an extremely ill patient had a nasal stomach tube in place for 17 days during which time a sore throat and pain in the neck and ears developed. Symptoms improved following removal of the tube, only to recur in 2 weeks and increase in severity to a degree requiring tracheotomy. This was done 1 month after removal of the feeding tube. Eventual subsidence of the process resulted in a chronic laryngeal stenosis for which a second tracheotomy had to be done to permit adequate treatment. At present she breathes freely without a tracheotomy and has a clear voice.

CASE 3. Mrs. E.A., secretary, white, 43 years of age. This patient had had four operations for gall-bladder disease between 1926 and 1940. In December, 1940, a fifth operation was performed and a nasal tube was left in place 7 days. The tube annoyed the patient considerably, causing a sore throat and considerable gagging, but the patient states she did not vomit. Symptoms improved following removal of the tube, but the sore throat became worse a week later, and gradually localized as severe pain deep in the neck, aggravated distressingly with swallowing. The patient developed hoarseness and then a complete aphonia 3 weeks later. Dyspnea began suddenly one night in February, 1941, and an emergency tracheotomy was done. Attempts to extubate her subsequently were unsuccessful. The patient was referred to St. Luke's Hospital in August, 1941, for laryngeal examinations. At this time the stenosis of the larynx was almost complete. The true cords could not be satisfactorily exposed because of marked anteroposterior compression of the larynx. Small bougies were

Fig. 7. Case 4. View of the hypopharynx, larynx, and esophagus from behind showing the ulceration of the postcricoid mucosa and exposure of the posterior surface of the cricoid cartilage. *A* Indicates the margin of the ulcer, *B* indicates the roughened posterior surface of the cricoid cartilage forming the base of the ulcer.

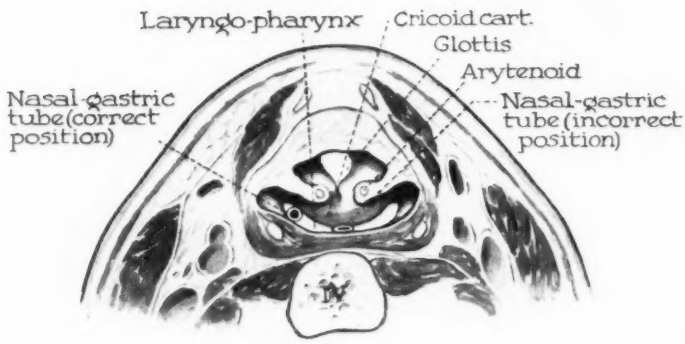

Fig. 8. Drawing of the horizontal section of the neck at the level of the 4th cervical vertebra and the cricoid cartilage showing the correct and incorrect positions of the feeding tube and the compression of the tube when in the incorrect position.

passed through what appeared to be the laryngeal aperture, but they met with considerable resistance. By means of laryngeal dilators a definite lumen was re-established and the patient could then phonate again by manually closing her tube. The examination demonstrated that the principal area of stenosis was immediately subglottic (Fig. 6). Dilatations were carried out at frequent intervals but the larynx never remained open sufficiently to permit removal of the tube. However, a valve tube was substituted for the original tube, and the patient is now able to speak well with the valve tube without manually closing the tube. At present she holds an important executive position and carries on all conversation without difficulty.

In this case the nasal stomach tube was in place 7 days and caused a sore throat and gagging, but no appreciable pain. After its removal, pain, hoarseness, and later aphonia developed. Approximately 6 weeks elapsed from the time the feeding tube was removed until the tracheotomy had to be done. The patient now has a chronic laryngeal stenosis and is wearing a valve tracheotomy tube. She refuses further surgery because she has a satisfactory functional result.

Case 4. A.M., 57 year old white female entered the Research and Educational Hospital on April 16, 1940, complaining of lower abdominal pain of 8 weeks' duration and persistent vomiting of 3 weeks' duration. The abdomen was distended with fluid, and rectal examination revealed a hard, fixed mass anterior to the rectum. Tumor cells were found in fluid removed by abdominal paracentesis. An exploratory laparotomy done 3 days after admission demonstrated an extensive carcinomatosis. A gastroenterostomy was done because of almost complete obstruction of the pylorus by the tumor. A stomach tube had been inserted when the patient entered the hospital and it remained in place for 5 days. It was then removed, and reinserted three times during the following 6 days; it was always tolerated poorly. The entire surgical wound opened on the eighth postoperative day. Reinsertion of the stomach tube at this time was associated with considerable trauma. The tube was withdrawn in an hour because gastric contents were not recovered. The overzealous attempt to insert the tube resulted in a perforation of the esophagus and the patient expired 3 days later of the combined effects of the abdominal and mediastinal pathology. The primary tumor was found to be a papillary cystadenocarcinoma of the right ovary with extensive metastases. Postmortem examination of the esophagus demonstrated a deep ulcer 4 centimeters in length in the mucosa covering the posterior aspect of the cricoid cartilage (Fig. 7). The ulcer extended through all the walls of the esophagus and exposed an area of the cricoid cartilage which then made up the base of the ulcer. The cartilage itself was roughened and necrotic in this area. A recent perforation of the left pyriform sinus was found which led into a necrotic mediastinal abscess. The postcricoid erosion seemed older than the perforation and was distal to it. It was undoubtedly present before the final attempts were made to reintroduce the tube.

While this patient did not have a true feeding tube stenosis of the larynx, the postmortem examination of the larynx demonstrates the earlier phase in the process which ultimately leads to the development of stenosis. The stomach tube was in place a total of 6 or 7 days, always poorly tolerated. The presence of the ulcer across the slightly convex posterior surface of the cricoid cartilage is significant in that the cartilage is thus exposed to the action of bacteria as well as to the chemical action of regurgitated gastric contents.

DISCUSSION

Various types of rubber tubes have long been used for emptying the stomach or for feeding purposes. The type known as the Levin tube is usually employed, as it is of smaller caliber and more pliable rubber than the larger tubes. The continuous suction technique using this tube, as employed by Wangensteen (6), has resulted in widespread use of the Levin tube for relatively long uninterrupted periods. Passed through the nose, it descends into the pharynx, the laryngopharynx, the esophagus

and the stomach. The only points of pressure throughout this course are in the nose, where the tube may become wedged between the turbinates and the septum, and in the postcricoid area of the laryngopharynx. At this point, the upper esophageal pinchcock at the entrance of the esophagus, the part of the inferior pharyngeal constrictors known as the cricopharyngeus muscle, keeps the cricoid cartilage pressed firmly against the cervical spine in an attempt to keep the lumen closed (Fig. 8). Both the posterior esophageal wall in this area (1) and the mucosa covering the cricoid cartilage are extremely thin, and consequently the thin mucous membrane affords almost no cushioning for these firm surfaces. When a feeding tube is inserted and left in place for several days or weeks, the pressure and rubbing action of the tube apparently produce ulceration of the postcricoid mucosa to allow infection to enter the body of the cricoid cartilage. Constant friction which occurs with swallowing, respiration, and speaking could augment the action of simple pressure on the cricoid cartilage produced by the tube. As Mosher suggests, this action is further increased by tension on the tube if its distal portion extends into the duodenum, and the gut actively attempts to advance the tube. The general nutritional condition of the patient is undoubtedly another factor, with the probability that ulceration occurs most frequently in debilitated patients. The destruction of the cricoid cartilage is further enhanced by chemical action if the patient vomits frequently. In Case 1 the acute episode of obstruction which precipitated the need for tracheotomy was preceded by 2 days of rather persistent vomiting. However, vomiting was not a prominent factor in the remaining cases.

Perichondritis of the larynx is a common clinical entity, but few cases due to erosions caused by feeding tubes have been presented. Those reported by Iglauer and Molt constitute the largest series. In the discussion of this report, 6 additional cases were presented. One other case was recorded by N. von Soubiron, G. Gerchunoff, and C. M. Emiliani. The earlier literature does not mention the feeding tube as a cause of laryngeal perichondritis. Morrison and Wangensteen have both mentioned that perichondritis and chondritis of the cricoid cartilage can occur, and Wangensteen has added 2 cases to the literature.

In reviewing the 4 cases herein presented, certain significant factors become apparent. All 4 were women. However, in the 11 cases presented by Molt and Iglauer there were 5 women, 4 men, 1 boy and 1 girl. Of the 2 recorded by Wangensteen one was a man and the sex of the second is not recorded. Three of our patients had laparotomies, the fourth (Case 1) had an abdominal pathological condition but her management was entirely medical. The tube was in place 4 days in Case 1, 17 days in Case 2, 7 days in Case 3, and a total of about 7 days in Case 4, continuously only 5 days. None of the 4 patients tolerated the tube well and all complained of pain and a sore throat. One patient, Case 4, died of causes other than those due to the feeding tube pathology. In the other 3 cases a tracheotomy had to be done: 43 days after the feeding tube was removed in Case 1, and 4 and 6 weeks after the tube was removed in Cases 2 and 3, respectively. In these 3 cases symptoms of pain, sore throat, and hoarseness subsided after the Levin tube was removed, only to recur gradually with increased intensity. The ultimate configuration of the larynx and the laryngeal pathology were similar in the 3 cases—destruction of the cricoid and thyroid cartilages, marked subglottic stenosis, and extreme limitation of motion of the vocal cords.

The symptom of pain associated with this condition is an important one and bears further analysis. The patient usually complains of a sore throat first. It is conceivable that a pharyngitis is necessary to initiate the sequence of pathologic changes that take place. It is just as probable that pharyngitis results as the mucosa first ulcerates and allows infection to enter the submucosal tissues. With inflammation and edema of the arytenoids pain radiates to the ears for the same reason that tuberculous laryngitis characteristically produces pain in the ears—namely because of referred pain carried there by the auricular branch of the vagus nerve.

TREATMENT

This discussion and presentation of cases should in no way be construed as a condemnation of the use of the indwelling stomach tube. However, if, during its use the patient complains of a sore throat or pain in the neck radiating to the ears, or if hoarseness is noted, the tube should be removed. If the condition of the patient does not permit removal of the tube, its position at the level of the larynx should be changed, noting that there is an actual change by visualization with the laryngeal mirror. It would appear likely that this type of ulceration and subsequent stenosis is not more common because in most instances the tube does not pass directly between the cricoid cartilage and the cervical spine, but descends laterally on either side of this point.

Active treatment of laryngeal perichondritis once it has developed is exceedingly difficult and often disappointingly ineffectual. Chemotherapy, both the sulfa drugs and penicillin, should be administered, although none of these patients was in the acute stage when penicillin was available and consequently no clinical experience can be recorded. A tracheotomy is indicated, of course, if respiratory embarrassment becomes severe. Surgical drainage and sequestration of the larynx and its cartilages should be postponed, if possible, rather than attempted early, because the cricoid cartilage is the only complete ring in the trachea, and its loss results in a collapse of the airway. No laryngeal instrumentation is done during the acute phase of this condition. Subsequently, however, active laryngeal dilatation or surgical reconstruction of the airway is necessary in order to remove the tracheotomy tube permanently. On occasion it is possible to remove the tracheotomy tube after the acute edema and perichondritis subside. Stenosis may recur in such cases several months or years later as the scar tissue contracts (Case 2).

SUMMARY AND CONCLUSIONS

Severe laryngeal destruction occasionally results from the routine use of nasal feeding or gastric suction tubes. Fortunately this complication is rare; however, it is so severe when it does occur that it is important to be aware of the possibility of this complication and to recognize its beginning symptoms at once.

Four cases are presented. In 3, rubber nasal stomach tubes were used, 4, 17, and 7 days, respectively, during which time the patients complained of discomfort due to the tube. Symptoms increased with sore throat, pain radiating into the ears, pain on swallowing, hoarseness, and finally dyspnea. Tracheotomies had to be done in these 3 cases 4 to 6 weeks after removal of the stomach tubes. All developed severe chronic laryngeal stenoses. One has been extubated, one wears a valve tracheotomy tube, and the third is still under treatment. The fourth patient died as the result of extensive carcinomatosis and mediastinitis. Postmortem examination demonstrated the ulcer which had not as yet caused laryngeal destruction.

A review of the literature reveals 24 recorded cases similar to those herein described.

Treatment consists of removing or changing the position of a feeding tube if the patient complains of a sore throat, and especially if hoarseness or pain in the ears develops. Active treatment, aside from chemotherapy, consists of tracheotomy, drainage, and subsequent laryngeal dilatation or plastic repair.

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THE PROTEIN NUTRITION OF SURGICAL PATIENTS

THAT the surgeon should be concerned with the chemical and physiological aspects of his patients is now such a well known fact that it is trite to mention it.[1] There is no form of tissue repair in which the protein metabolism is not concerned. Protein metabolism is also concerned with resistance to infection, and resistance to poisons of various kinds. Serious protein depletion of the body tissues may be present in a large proportion of patients, particularly those who are markedly under weight. Such depletion may be the result of either acute or chronic illness. An acute illness may cause protein depletion with great rapidity, particularly in diarrheal diseases, burns, and major infections. Even in simple major fractures, net losses of protein representing a pound of tissue a day may go on for weeks.

Fortunately, methods to prevent or reduce these net losses are becoming widely available even for patients who cannot eat. Following the pioneer work of Elman,[2] intravenous protein hydrolysates and mixtures of amino acids for intravenous use have been found to be safe and practical, but the latter are not yet available for general use.

Parallel with this development, improved products for oral and intubation feeding have also become available. As one becomes familiar with the results following the use of such substances, the indications for their use become broader. The time has come when surgeons should become thoroughly familiar with the protein requirements of the normal and of the sick individual. In thinking of such requirements, one must be careful to think of the requirements in terms of optimum use rather than in terms of the requirements for bare existence. When time is available to improve protein nutrition before surgery, and when this time is used efficiently for this purpose, the reduction in postoperative shock and other complications is impressive. Under certain conditions indicated in a very suggestive paper of CoTui[3] on gastroduodenal ulcers, improved protein nutrition may even abolish the necessity for surgery. When conditions do not allow time for preoperative repair of protein deficiency, postoperative repair at the earliest moment is very important.

Protein hydrolysates are not an efficient treatment of shock and should not be used during threatened or actual surgical shock. Whole blood is the method of choice in the treatment of this complication, with plasma

[1]Lund, C. C., and Levenson, S. M. J. Am. M. Ass., 1945, 128: 95–100.

[2]Elman, R. Ann. Surg., 1940, 112: 594–602.

[3]CoTui, Wright, A. M., Mulholland, J. H., Galvin, T., Barcham, I., and Gerst, R. Gastroenterology, 1945, 5: 5–17.

and human albumin as effective agents to be used temporarily. Immediately after the danger of shock is over, protein hydrolysate or food may be given.

Plasma is being widely used in an attempt to repair protein deficiency. This is not an efficient method of treatment for this purpose. Whether money has to be paid for plasma or not, by the hospital or by the patient, plasma is a very expensive material. It has been calculated that the protein in plasma costs at least one dollar per gram. The corresponding price for lean hamburg steak in a generous serving of ⅓ of a pound would be about twenty-five dollars. As a treatment for hypoproteinemic patients, one must also remember that plasma contains a great deal of sodium in the form of the original sodium chloride in the blood plus a generous amount of sodium in the form of citrate added to prevent clotting. When plasma is used in large quantities in the patient with true hypoproteinemia, increased edema and heart failure may well result.

Under conditions of severe burns or severe depletion from various infections or intestinal disabilities up to 400 or 500 grams of protein per day may be life-saving. Up to 150 grams per day of protein can be given intravenously in the form of hydrolysates or other similar preparations. Glucose may be and should be added to these hydrolysates because of its protein sparing effect. A formula suitable for many conditions has been suggested by Butler[1] and consists of 1 liter of amigen plus 300 cubic centimeters of 50 per cent glucose. This cannot be given in less than 4 or 5 hours because rapid administration causes nausea and vomiting, and if given into a moderate sized vein, seldom causes thrombosis. Three such injections a day will furnish 150 grams of protein and 600 grams of carbohydrate. This amount of food represents 3000 calories.

Experience has shown that vitamins should be added to such a mixture in generous amounts for the patients who are sick enough to warrant this treatment. There is apparently very little wastage if 1 gram of ascorbic acid, 200 milligrams of niacin, 20 milligrams of riboflavin, and 20 milligrams of thiamin are given to such a patient at some time during each 24 hours.[2] Oral feeding or tube feeding to the hypoproteinemic patient or the patient in whom efforts are being made to avoid hypoproteinemia, should be in the form of mixtures of protein and carbohydrates with a minimum of fat. The following formula has been taken over periods of months by patients with burns which were so extensive that early grafting of the whole area was impossible.

HIGH PROTEIN MIXTURE FOR ORAL FEEDING

		Cbh.	Prot.	Fat	Calories
Skim milk	3.0 L	150	110	6	1094
Skim milk powder	300 G	117	90	6	828
Amigen powder	100 G	0	75	0	600
Valentine's liver extract	30 cc.	0	2	0	8
Salt	15 G	0	0	0	0
		267	277	12	2530

This mixture makes up a large part of the food necessary for 24 hours and should be given in 8 ounce portions every two hours day and night. The patient taking this will have little appetite for any other food, but meat and carbohydrate food may be given immediately after at least three liquid feedings. Such a regimen avoids the very serious difficulty of the patient with a poor appetite choosing the more palatable but less important nutritional elements from an ordinary high protein diet tray and gives the

[1]Butler, A. M., and Talbot, N. B. N. England J. M., 1944, 231: 621-628.

[2]Levenson, S. M., Green, R. W., Taylor, F. H. L., Robinson, P. Page, R. G., Johnson, R. E., and Lund, C. C. Ann. Surg. (in press).

surgeon a better degree of control over the patient's intake than any previous method of feeding. When such feeding is used for any condition other than burns or other than conditions where chronic salt depletion has been demonstrated, the amount of added salt should be greatly reduced or eliminated entirely. When a patient cannot or will not take this diet, tube feeding may be indicated and such a mixture may have powdered yeast and more amigen added because palatability is not a factor. A suitable formula for this consists of:

HIGH PROTEIN MIXTURE FOR TUBE FEEDING

		Cbh.	Prot.	Fat	Calories
Skim milk	3.0 L	150	110	6	1094
Skim milk powder	200 G	78	60	6	598
Amigen powder	200 G	0	150	0	600
Valentine's liver extract	30 cc.	0	2	0	8
Brewer's yeast pwdr.	30 G	12	14	1	113
Salt	15 G	0	0	0	0
		240	336	13	2413

The patients who need these types of oral or tube feeding also need vitamins, but over a long period of time probably do not need as high a dose of vitamins as the patients who are receiving parenteral protein feedings. Suggested oral daily doses for these patients would be vitamin A, 30,000 units; vitamin D, 3,000 units; ascorbic acid, 500 milligrams; thiamin, 20 milligrams; riboflavin, 12 milligrams; and niacin, 100 milligrams.

Charles C. Lund.

CONGENITAL HYPERTROPHIC PYLORIC STENOSIS

THE past quarter century has shown remarkable progress in the surgical management of most abdominal conditions and much of this seems to be due to our increased understanding of what Cannon has referred to as homeostasis. The surgeon is now primarily an excellent physiologist and only secondarily a technician. The time and care which we all devote to the nutritional and electrolytic requirements of the body, both preoperatively and postoperatively, forcefully attest this fact.

Nowhere is this progress more clearly reflected than in the modern treatment of congenital hypertrophic pyloric stenosis, because nowhere else do we find such an extreme lability of this homeostatic mechanism. Man, who may be considered the most competent animal, produces the most helpless young, and this helplessness extends all the way from inability to avoid physical danger to inability to maintain a stable internal environment except under the most favorable circumstances. Obviously, therefore, when an infant of two or three weeks of age begins to vomit everything taken by mouth it will be but a short time before extreme dehydration and loss of electrolytes will so disrupt the delicately balanced biochemistry of his body that continuance of life will become endangered. While these facts are now so evident that to repeat them is almost tautological, it was but a few years ago that these acutely ill, dehydrated babies were subjected to emergency surgery. The surprising fact is that the operative mortality was only 35 to 40 per cent.

It seems to me that we were all very slow in realizing that these babies needed emergency "physiology" rather than surgery. They were desperately, albeit weakly, crying to us to replace their fluids and electrolytes so that they might survive this crisis which was threatening their existence. Having finally awakened to an understanding of these biochemical disturbances and having taken proper steps to aid the infant to correct them, it soon became obvious that the operation could be delayed several days, if necessary, until hydration was accomplished. Lowered mortality resulted.

At first these infants were given blood transfusions routinely as preoperative preparation. Later it was found that the parenteral administration of physiological solutions of glucose and saline produced the same result with fewer complications and less expense. Transfusions were then reserved for the infants which showed advanced dehydration, and were used in conjunction with the parenterally administered electrolyte solutions. Increased experience in this direction produced a further decrease in mortality.

Another factor which has improved the outlook for these babies has been the more prompt recognition of the disease. The typical and almost constant history of the infant who does very well for the first two weeks of life, then begins to regurgitate part of his feedings, then to vomit the whole feedings and finally to develop the characteristic projectile vomiting of bile free fluid, has become so well known to the medical profession that these babies are sent immediately to the hospital. They therefore arrive in much better condition and frequently they can be prepared in two or three days for surgical cure.

Usually, on arrival at the hospital, they are dehydrated, constipated, pass only small amounts of concentrated urine, and show visible peristaltic waves travelling across the abdomen. Palpation, especially when the stomach is empty, will reveal the pyloric tumor which is pathognomonic of the disease. Restoration of body fluids, electrolytes and, if necessary, serum proteins, is promptly begun, and, when restoration has been accomplished as demonstrated by clinical observation and the judicious use of laboratory tests, with probably major emphasis on the former method, surgery should be undertaken at that time.

The ultimate cure in every case seems to lie in the Fredet-Rammstedt operation, because it gives a permanent result and is very simple and rapid in its execution. We prefer a right rectus incision placed so high that the right lobe of the liver must be displaced upward to expose the pylorus. Great care must be used in dividing and spreading the hypertrophied pyloric musculature so that no damage to the underlying mucosa occurs. After careful layer-by-layer closure of the abdomen, the healing incision being effectively buttressed by the liver, we are reasonably insured against postoperative wound dehiscence.

After operation the baby should be kept in a room where the temperature is constant and where he is completely isolated from all other cases. Everyone who enters the room should wear a clean gown and mask. Extreme gentleness in handling the baby and the use of a small cane sugar pacifier at the time of wound dressings are of course, mandatory.

The surgical mortality in this condition at the present time is about 1.2 per cent. We are convinced that with proper preoperative, operative, and postoperative care, mortality should be about zero as far as the operation is concerned. Because the surgical mortality and morbidity are so low, we never consider prolonged medical treatment such as refeeding after vomiting, feeding by gavage, etc. The facts that ten days after this operation the baby is gaining weight and hungrily taking a generous evaporated milk formula and, that within three to four weeks, he has regained the position he would have held had he never had pyloric stenosis are convincing evidence of the efficacy of the operative method in the hands of the surgeon who will first carefully treat the baby and then skillfully treat the disease.

EDWARD J. DONOVAN

THE SURGEON'S LIBRARY

REVIEWS OF NEW BOOKS

AS may be judged from the title, the two volume, 1200 page work *Surgical Treatment of the Motor-Skeletal System* by Bancroft and Murray[1], embraces the entire field of traumatic and non-traumatic orthopedic surgery. While created and fostered by the Division of Orthopedic Surgery at Columbia University, the collaborators number 43 men whose individual work and writings have made them outstanding in the fields of their special interests.

The majority of the authors are from the Eastern schools and clinics, but the overall impression is gained that no personal, group, or sectional viewpoint is stressed. Certain principles and methods of treatment are, of course, emphasized, because the author, in a number of instances, has originated them or has been responsible for their wide acceptance. For example, it would be expected that Dr. von Lackum would present the subject of scoliosis as he does, for much of our present management of this condition comes from the work of the New York Orthopedic Hospital Staff. Dr. Kite, who has made an art of the wedged cast treatment of foot deformities, naturally devotes more space to this method than to others. It is to be regretted that some of the articles were completed before the newer concepts of chemotherapy were generally accepted, but the editors have overcome this to some extent, in that they have written on chemotherapy as it applies to all the infections attacking the skeletal system. An example of this may be found in Dr. Beekman's chapter on osteomyelitis. This chapter is also an example of the thoroughness with which various conditions are discussed. Dr. Beekman draws on his wide experience in the Children's Surgical Service at Bellevue Hospital, so that an excellent background on osteomyelitis can be obtained by reading this chapter. Some of the newer concepts, more or less experimental at the time this work was completed, can be found in the section on chemotherapy.

It was somewhat disappointing to find nowhere in the book a chapter dealing entirely with the infections, injuries, and other conditions of the hand. One interested in looking up something in this field would be forced to seek through many chapters before finding the specific information required. The chapter entitled "Military Surgery" adds little to the usefulness of the book, but of course one may suggest that most of the important work that came out of the war is included other places.

The general scheme of the volumes suggests that much thought was given to the presentation plan and this, plus the unusual activity of Dr. Murray, whose own views are many times expressed aptly if not sharply, makes it a fully adequate survey. The presentation of each subject is followed by a brief but comprehensive bibliography containing the citation of really important work that has been done in that particular field. Most reviewers will agree that there is a uniformity and smoothness of style throughout which is not found in the usual medical text by multiple collaborators, and this absence of encyclopedic jerkiness will be an incentive to become cover-to-cover readers of the two volumes. This was true of the undersigned, and it is something he has seldom before done.

It is impossible to discuss in detail all of the many chapters. The man interested in a particular problem at hand is sure to find complete and authoritative information. On the other hand the student, whose task it is to read for general rather than specific information will find his needs well filled. I know of no two volumes available now that would be more useful in the school years or afterward than this splendid work.

JAMES K. STACK.

EDITED by W. G. Barnard and A. H. T. Robb-Smith *Kettle's Pathology of Tumours*[2] is a most concise treatise. Since it was written for students, it is a didactic presentation with but little argumentation and few references to source material. Present day ideas in tumor fundamentals are adhered to generally. The development of this third edition (1946) is shown most strikingly if one compares certain sections in it with those of the first edition (1916). The sections on etiology, tumors of the lungs, and tumors of the nervous system have altered so much in these 30 years that they are entirely new concepts. Also in keeping with present times is the inclusion of a section on trauma and on occupation in relation to tumors. The book is richly illustrated by nearly 200 drawings and photomicrographs in approximately 300 pages of text.

HAMILTON R. FISHBACK.

THE two pocket books *Lecciones de cirugía*[3] are the first of a series to appear in the future. They are intended to give a written account of the Postgraduate Surgical Instruction as carried out on the

[1]SURGICAL TREATMENT OF THE MOTOR-SKELETAL SYSTEM. Edited by Frederic W. Bancroft, A.B., M.D., F.A.C.S., and Clay Ray Murray, M.D., F.A.C.S. Parts 1 and 2. Philadelphia, London, Montreal: J.B. Lippincott Co., 1945.

[2]KETTLE'S PATHOLOGY OF TUMOURS. By W.G. Barnard, F.R.C.P., and A. H. T. Robb-Smith, M.A. (Oxon.), M.D. 3d ed. London and New York: Paul B. Hoeber, Inc., 1946.

[3]LECCIONES DE CIRUGÍA. By Ricardo Finochietto. 1st and 2d series. Buenos Aires: La Prensa Medica Argentina, 1945.

service of Dr. R. Finochietto at the Hospital Rawson.

Each surgical paper deals with a case. The indications as to the type of operation are first discussed, and the selected procedure is given in full detail with numerous highly informative comments. The series will form an excellent source of information as to how surgery is practiced routinely by Dr. Finochietto and his resourceful coworkers.

Páginas de cirugía[1] is a 262-page book and is dedicated to Dr. E. Finochietto by the same workers mentioned previously in Dr. R. Finochietto's service. Fifteen different surgical procedures, including all types of surgery, are described. These works appeared in the *Prensa medica Argentina* (August 9, 1944) and are presented herein in a book form. F. F. Remy.

IT is impossible to review Cantor's *Ambulatory Proctology*[2] without being moved to comment on the author's viewpoint, highlighted as it is in title and preface. The author advocates and urges the performance of such procedures as extensive hemorrhoidectomy, repair of rectal prolapse and fistulectomy (to mention only three of many operations listed) on an office ambulatory basis. This attitude of mind which takes cognizance of the mechanical and physical equipment of the doctor's office, rather than the hazard to the patient, is unsurgical, unscientific, and dangerous to say the least. The practitioner would do well to pause and think of his patient before embracing such frankly dangerous practice.

If one does not take too seriously the author's mode of practice, the material encompassed in this volume is commendable. The discussion of the various proctologic ailments, though limited chiefly to management, is well presented. Steps to be taken in treatment are listed in definite sequence. There is a good section on pediatric proctology, which is missing in most recent books on the subject. Too great emphasis is placed on "tattoo" technique or what the author refers to as "tattoo neurotomy" in the treatment of pruritis ani, a method by no means applicable to a very large percentage of cases and certainly not available or practical for the average doctor.

There are many good illustrations. Especially well done are those descriptive of fistula in ano and fistulectomy.

One half of the book is devoted to medical diseases of the rectum and colon. Each chapter is concluded with an up-to-date and extensive bibliography.

Thomas J. Merar.

THE book *Surgical Treatment of the Nervous System*[3] edited by Bancroft and Pilcher is very satisfactory, for it is simple; the descriptions are brief but adequate; and the twenty chapters have been contributed by seventeen different authors, so that there is considerable diversity. The plain truth is that neurosurgery, though a young and specialized field, does not lend itself well to an entire volume by a single author, for already among neurosurgeons themselves, there is some tendency to specialization; or, at least, their talents and interests lie in different directions. Thus, one clinic may see large numbers of protruded discs, another, many sympathectomies, and still another may largely occupy its time with tumor surgery. And no civilian neurosurgeon can hope to match the sophistication of the younger men with army experience, with respect to peripheral nerves and cranioplasty.

Dr. Pilcher has picked leaders in all the fields he has covered. Their attacks on the subjects assigned to them have been orthodox and sound. While the book is perfectly understandable to the beginning student in the field, it is also of interest to the most experienced. It is reasonably well illustrated, and this reviewer found it as competent a book as exists on neurosurgery. Eric Oldberg.

THE book *Modern Management in Clinical Medicine*[4] by F. Kenneth Albrecht is quite different from any of the usual textbooks of medicine, and is also different from the usual compendium of therapy. It attempts, and in great part succeeds, in being a handbook of the practice of medicine. It has an excellent professional attitude. Emphasis is placed upon the importance of a good case history as a basis for diagnosis, and each section contains a very complete printed case history form directed to the development of the histories typical of each system being studied.

The second chapter covers nutrition and vitamin deficiency diseases. It is brief, but complete and well illustrated. The unit standardization of all clinically available vitamins is given. As is true throughout the book, one might differ with particular stunts in treatment that appeal to the author, such as the use of benzedrine in the management of obesity.

The next chapter is on disorders of the digestive tract. Valuable and demonstrative x-ray pictures are reproduced, showing all important organic pathology of the gastrointestinal tract. Anorectal conditions are not neglected. This attention to such common aggravating, minor clinical conditions that make up such an important part of office practice is characteristic of this work, and will go far to make it of great usefulness.

The next main section of the book is on diseases of the blood and blood-forming organs by Dr. Seward E. Miller, Surgeon, U.S. Public Health Service, Chief of the Department of Pathology, U.S. Marine Hospital, Baltimore. This opens with a good modern discussion of various analyses in hemocytometry.

[1] Páginas de cirugía. By Enrique Finochietto. Buenos Aires: La Prensa Medica Argentina, 1945.

[2] Ambulatory Proctology. By Alfred J. Cantor, M. D. With a foreword by Beaumont S. Cornell, M. D. New York and London: Paul B. Hoeber, Inc., 1946.

[3] Surgical Treatment of the Nervous System. Edited by Frederic W. Bancroft, A.B., M.D., F.A.C.S. and Cobb Pilcher, M.D., F.A.C.S. Philadelphia, London, Montreal: J. B. Lippincott, 1946.

[4] Modern Management in Clinical Medicine. By F. Kenneth Albrecht, M.D. Baltimore: The Williams & Wilkins Co., 1946.

The actual commercial extracts of liver are listed, with their U.S.P. units.

The chapter on cardiology is extensive and includes a simple and useful discussion of electrocardiography. It is interesting that the use of testosterone propionate in the treatment of angina pectoris is recommended. There is a good chapter on peripheral vascular disease. Buerger's exercises are well illustrated. The treatment of varicose veins by injection is described in detail.

The discussion of diseases of the kidneys quotes completely and verbatim the excellent instructions of Dr. George Thorn for the management of various types of renal insufficiency. As one might expect from the U.S. Public Health Service, the diagnosis and management of venereal diseases has a long, very well illustrated and excellently detailed account. The colored illustrations of skin lesions are excellent.

The chapter on diseases of the respiratory system emphasizes physical findings, but also has an abundance of excellent x-ray reproductions. Pneumothorax treatment of tuberculosis is described in detail.

The chapter on arthritis, as usual, opens with an arthritis case history form. The treatment of rheumatoid arthritis is divided in an interesting manner into those measures of proved value, those which have had optimistic recommendations but of which the value is still undetermined or inconclusive, and those which are of questionable value or are useless.

The section on endocrinology for the general practitioner is of excellent practical value. The discussion of the management of diabetes is simple, very practical, and correct. In this field one might again differ as to a particular procedure, such as the permission to allow excessive thyroid dosage in the treatment of any case of myxedema; for instance, "2 grains three times daily for five days"; or to state "the treatment of myxedema is aimed at elevating the basal metabolic rate." The use of thiouracil is discussed, but the dangers of its administration are not sufficiently stressed. More emphasis in this chapter should be placed upon the treatment of the menopause. A useful table of commercial endocrine products with the trade name, the chemical name and the standardization of dosage is given.

The section on allergy is good. It contains a pollen table arranged according to months and states throughout the United States, and directions for desensitization management.

Dr. Miller contributes a useful chapter on toxicology, with a table of a great many poisons with their antidotes.

The infectious diseases are covered with the use of excellent illustrations, and a full discussion of the newest chemotherapeutic agents.

Tropical diseases of post war importance receive for the first time in an American textbook of medicine an adequate, thoroughly illustrated chapter.

There is a long section, as is deserving, on nervous and mental diseases, which opens with the statement: "The idea that the diagnosis of organic disease of the nervous system can be made only by those specializing in neurophysiology, neuroanatomy and neuropathology is too generally accepted by many intelligent practitioners." The use of the well illustrated and complete section which follows should go a long way to change this conception. Mental disorders are approached in an objective manner with an excellent outline of a "neuropsychiatric and psychosomatic case history record." Practical methods of psychiatric examination and modern methods of treatment are then discussed. It is a pleasure to find a practice of medicine including such a useful treatise on the practice of neuropsychiatry.

Again, as one would expect from the U. S. Public Health Service, the diagnosis and treatment of common skin disorders is given full and useful representation.

Chemotherapy and antibiotic therapy are covered in Chapter 18. In harmony with the changing practice of medicine, geriatrics, the care of the aged, receives a chapter of its own. A chapter containing many useful hints for the general practitioner in his important duty of managing the patient at home rather than in the hospital is covered in a chapter labeled "The Care of the Ambulatory Patient."

Finally Dr. Miller adds a short compact chapter on clinical laboratory methods, including that bête noir of the general practitioner, the listing of chemical values in milliequivalents per liter rather than in milligrams per 100 cubic centimeter. Not satisfied with such complete coverage, the author adds an appendix covering directions for the performance of many "common procedures," such as thoracentesis, pericardial and abdominal paracentesis, blood typing, and blood transfusion, hydrotherapy, genitourinary treatment, and a list of practical diet menus.

Each of the twenty-one chapters and appendix is followed by a short, but authoritative list of modern references.

In summary one may say that the internist and general practitioner will find this a very helpful and useful work.

PAUL STARR.

The small volume *Motor Disorders in Nervous Diseases* by Ernst Herz[1] was first prepared as a syllabus to be used together with a collection of teaching motion picture films on the general subject of neurology. Alone, with the many good illustrations furnished, it is actually a descriptive atlas dealing with the testing of motor function and the commoner forms of the pathology of motion.

This book should be a valuable aid to students who are struggling to apply their usual maze of anatomical and physiological facts to the study of actual disease states in patients, since both the illustrations, some of which are good diagrams, and the straightforward and uncomplicated text put the disorders of motor function on a practical, physiological basis. It is pointed out that a large part of any

[1]MOTOR DISORDERS IN NERVOUS DISEASES. By Ernst Herz, M.D., and Tracy J. Putnam, M.D. New York: King's Crown Press, 1946.

complete neurological examination is concerned with a study of the motor function, and the book proceeds according to a plan which the authors believe to be a logical approach in clinical diagnosis. The common reflexes are shown in photograph form. Such terms as *apraxia*, *dystonia*, and *tic* are illustrated by excellent figures and described in simple, brief, understandable text. Almost half the book is concerned with the motor disorders of the cranial nerves, by no means a disproportionate amount of space for that part of neurological study which is so often confusing to the beginner.

This is not an exhaustive treatise. Its primary purpose is that of use with prepared motion pictures. Nevertheless it is, alone, a book that will be found useful to students and teachers alike. Perhaps the authors will decide to publish a companion text on sensory dysfunction and other special phases of neurological examination.

The book is published by the King's Crown Press, "a division of Columbia University Press organized for the purpose of making certain scholarly material available at minimum cost." The modest price of the book is three dollars. JOHN MARTIN.

BOOKS RECEIVED

Books received are acknowledged in this department, and such acknowledgment must be regarded as a sufficient return for the courtesy of the sender. Selections will be made for review in the interests of our readers and as space permits.

DEMONSTRATIONS OF OPERATIVE SURGERY FOR NURSES. By Hamilton Bailey, F.R.C.S. Baltimore: Williams & Wilkins, 1945.

SYNOPSIS OF OBSTETRICS AND GYNAECOLOGY. By Aleck W. Bourne. 9th rev. ed. Baltimore: Williams & Wilkins Co., 1945

PNEUMOPERITONEUM TREATMENT. By Andrew Ladislaus Banyai, M.D., F.A.C.P., F.C.C.P. St. Louis: The C. V. Mosby Co., 1946.

A TEXTBOOK OF GYNECOLOGY. By Arthur Hale Curtis, M.D. 5th ed. Philadelphia and London. W. B. Saunders Co., 1945.

AUTOPSY DIAGNOSIS AND TECHNIC. By Otto Saphir, M.D. With a Foreword by Ludvig Hektoen, M.D. 2d rev. enl. ed. New York, London: Paul B. Hoeber, Inc., 1946.

THE RESULTS OF RADIUM AND X-RAY THERAPY IN MALIGNANT DISEASE. 2d Statistical Report from The Holt Radium Institute, Manchester, 1934-1938. Edinburgh: E. & S. Livingstone, Ltd., 1945.

BABY; A MOTHER'S MANUAL. By Stella B. Applebaum. With introductions by Bela Schick, M.D., and Margaret E. Fries, M.D. Chicago, New York: Ziff Davis Publishing Co., 1946.

DIAGNÓSTICO Y TRATAMIENTO DE LAS VARICES ESENCIALES. By J. Valla-Serra. Barcelona: Librería de Ciencias Médicas, 1945.

THE MODERN TREATMENT OF DIABETES MELLITUS. By William S. Collens, B.S., M.D., and Louis C. Boas, A.B., M.D. Springfield: Charles C. Thomas, 1946.

SPEZIELLE CHIRURGISCHE THERAPIE. By Dr. Max Saegesser. Bern: Hans Huber, 1945.

DISEASES OF THE RETINA. By Herman Elwyn, M.D. Philadelphia, Toronto: The Blakiston Co., 1946.

HUMAN TORULOSIS. By Leonard B. Cox., M.D., M.R.C.P., F.R.A.C.P., and Jean C. Tolhurst, M. Sc. Melbourne: Melbourne University Press, 1946.

ANESTHESIA IN GENERAL PRACTICE. By Stuart C. Cullen, M.D. Chicago: The Year Book Publishers, Inc., 1946.

MEDICAL EDUCATION AND THE CHANGING ORDER. By Raymond B. Allen, M.D., Ph.D. New York: The Commonwealth Fund, 1946.

LA MÉTHODE DU LEVER PRÉCOCE EN CHIRURGIE ABDOMINALE. By André Chalier. Paris: Masson et Cie, Editeurs, 1945.

WOMEN IN INDUSTRY; THEIR HEALTH AND EFFICIENCY. Issued Under the Auspices of the Division of Medical Sciences and the Division of Engineering and Industrial Research of the National Research Council. By Anna M. Beatjer, Sc.D. Philadelphia and London: W.B. Saunders Co. 1946.

CLINICAL CONGRESS OF AMERICAN COLLEGE OF SURGEONS

W. Edward Gallie, Toronto, *President*
Irvin Abell, Louisville, *President-Elect*

Committee on Arrangements
Howard A. Patterson, *Chairman;* Frank Glenn, *Secretary*

CLINICAL CONGRESS IN NEW YORK POSTPONED BECAUSE OF UNITED NATIONS ASSEMBLY

The United Nations General Assembly which will be held in New York this September has made it necessary to postpone until 1947 the local features of the following proposed program in New York. The material was ready for publication when it became necessary to cancel the 1946 Clinical Congress in New York and it is therefore being published herewith without change. Plans are now under consideration for a meeting to be held this fall in another city where adequate accommodations might be available.

NINETY days after arrangements had been completed to hold the thirty-second annual Clinical Congress of the American College of Surgeons in New York from September 9 to 13, plans were announced for the United Nations Assembly in that city during the same month requiring hotel accommodations for around six to eight thousand people. Acting upon authoritative advice, the College at first proceeded with plans for the Congress, and the Committee on Arrangements in co-operation with the local hospitals drew up a most excellent clinical program which is published in succeeding pages. Later developments made it increasingly evident, however, that the Fellows would be put to so much discomfort and inconvenience, if indeed they were able to obtain hotel accommodations at all, that postponement of the Clinical Congresss was imperative.

The decision to postpone the Congress was made with especial reluctance because of the fact that no later date this year seems to be available for the meeting in New York. Negotiations are in progress and it is hoped that announcement of a definite date can be made in the very near future. Because of the well advanced stage of the program for both the clinical and scientific sessions, and the superior facilities available in New York for the momentous event which the next Congress will be after so long a period without annual meetings, it is not considered desirable to move to another city unless absolutely necessary. Therefore, work on the program in New York is proceeding, in accordance with the outline in the following pages.

SIXTH NEW YORK CONGRESS

This will be the sixth time that the Clinical Congress has been held in New York. The first time was in 1912, when the old Waldorf-Astoria was the scene of the third Congress. Dr. George E. Brewer was the Chairman and Dr. Charles H. Peck the Secretary of the 1912 Committee on Arrangements. Dr. Edward Martin of Philadelphia was then President of the Clinical Congress of Surgeons of North America. More than 2,600 physicians registered, and the lack of organization for regulating the attendance at the clinics precipitated the proposal by Dr. Franklin H. Martin at the business meeting on Friday afternoon, November 15, for the creation of the college.

At the same meeting Dr. Allen B. Kanavel presented a resolution which provided that "some system of standardization of hospital equipment and hospital work should be developed, to the end that those institutions having the highest ideals may have proper recognition before the profession, and that those of inferior equipment and standards should be stimulated to raise the quality of their work. . . ." and that "the President of the Congress be authorized to appoint a committee from the profession, delegated to carry the spirit of this resolution into effect, and report at the Clinical Congress in 1913."

Another significant event was the appointment, during an evening session given to the consideration of cancer, held in Brooklyn, of a committee that formed the nucleus of the American Society for the Control of Cancer which was organized following the Congress of Physicians and Surgeons of North America on May 22, 1913.

Thus the plans for formation of the College and for the inauguration of activities in two fields which have always been among its major concerns, hospital betterment and cancer control, were initiated at the 1912 Clinical Congress in New York, 5 years before the Congress was amalgamated with the American College of Surgeons. Later Congresses were held in New York in 1919, 1924, 1931, and 1938.

FIRST POSTWAR CONGRESS

The first postwar Clinical Congress will differ from the meetings for several years prior to the war in that a return will be made to the original custom of holding the Presidential Meeting on the opening evening, Monday, and the Convocation on the final evening, Friday, instead of combining them on the first night. There are several good reasons for the change among which is the likelihood of having several hundred surgeons, who have been received into fellowship *in absentia* during the years in which no Convocation was held, added to the number of initiates for the current year who will be present for the initiation ceremonies. Related to this is the need for relieving the pressure for hotel accommodations by distributing the attendance better throughout the week than would be possible if the Convocation were held on the opening night.

COMMITTEE ON ARRANGEMENTS

A representative Committee on Arrangements has been appointed consisting of the following members:

Howard A. Patterson, *Chairman*	George J. Heuer
Frank Glenn, *Secretary*	J. William Hinton
Frank E. Adair	Virginius B. Hirst
Albert H. Aldridge	George H. Humphreys
Thomas M. Brennan	Robert H. Kennedy
E. Jefferson Browder	William F. MacFee
George F. Cahill	John H. Mulholland
Henry W. Cave	W. Barclay Parsons
Ralph Colp	Brittain F. Payne
Edward J. Donovan	Otto C. Pickhardt
Merrill N. Foote	Thomas H. Russell
John H. Garlock	Raymond P. Sullivan
Charles A. Gordon	Howard C. Taylor, Jr.
	William Crawford White
	Philip D. Wilson

The Committee has been working diligently for several weeks on plans for the meeting.

CLINICAL PROGRAM

The hospitals and medical schools of greater New York are co-operating in scheduling operative and nonoperative clinics, and group clinical conferences. Visiting surgeons will have ample opportunity to attend well arranged programs of many different kinds in several of the excellent hospitals in the area. General and special demonstrations will be held such as fractures, cancer, maternal morbidity, and end-result studies; clinicopathologic and x-ray conferences; newer diagnostic and therapeutic procedures; preoperative and postoperative supportive treatment; anesthesia; and reconditioning. The newer techniques and surgical procedures may be observed by those who are interested in them.

The medical schools will hold a series of exhibits demonstrating their work which will be on display in their affiliated hospitals. The program of each hospital will be arranged to cover subjects in general surgery, obstetrics and gynecology, fractures, orthopedic surgery, thoracic surgery, neurosurgery, genitourinary surgery, ophthalmology, and otolaryngology.

PRESIDENTIAL MEETING

On Monday evening in the Grand Ballroom of the Waldorf-Astoria, the Presidential Meeting will be opened with the impressive processional of the officers, regents, and honorary guests. Welcome to the assembly will be extended by the chairman of the Committee on Arrangements, Dr. Howard A. Patterson. Dr. W. Edward Gallie of Toronto, President of the College, will preside and will deliver the Presidential Address. An inaugural ceremony will be held for the incoming officers. Foreign guests will then be introduced.

A new feature to be included in the Presidential Meeting this year will be the first Martin Memorial Lecture. Dr. Edward D. Churchill of Boston has accepted the invitation to give this lecture. This lectureship was established upon motion of the Board of Regents at its midyear meeting on April 1, as a memorial to both Dr. Franklin H. Martin and Mrs. Martin. The founder of the College and of SURGERY, GYNECOLOGY AND OBSTETRICS was joined by his wife in making the College the beneficiary at their deaths of the Journal, together with its physical plant operated by the Surgical Publishing Company of Chicago, of which they were the owners. Doctor Martin died in 1935 and Mrs. Martin in 1945.

The Martin Memorial Lecture to be given annually during Clinical Congress, and dealing with a scientific subject of the author's choosing, will supplant the former Annual Oration in Surgery.

CONVOCATION

It is planned this year, for various reasons, to depart from the custom of recent years of holding the Presidential Meeting and the Convocation on the same evening. The Convocation is therefore scheduled for Friday evening. An Assembly of Initiates will be held prior to the Convocation on Friday in order to instruct them in the Convocation procedure and other matters.

The Convocation will open with a processional of officers, regents, and governors. The President, Dr. Irvin Abell, will preside and will make the opening remarks, confer the honorary fellowships, and present the candidates for fellowship. Following this ceremony, a speaker to be announced later will give the Fellowship Address.

FORUMS ON FUNDAMENTAL SURGICAL PROBLEMS

The Forums on Fundamental Surgical Problems will be conducted on Tuesday, Wednesday, Thursday, and Friday mornings. Included in them will be brief reports of original clinical and experimental observations relating to the broad aspects of surgery and the surgical specialties. No prepared discussions of the reports are planned, but questions and comments will be invited. Especially keen interest is expected in these sessions this year because of the accumulation of the results of 5 years of work since the last Clinical Congress. Dr. Owen H. Wangensteen of Minneapolis, chairman of the committee which is planning the program, is working toward representation of as many as possible of the various university departments of surgery in this presentation of clinical and experimental research work.

The enlistment of the interest of young men who are doing original work, through the forums, is one of the most beneficial results of these sessions which are now considered to be an indispensable feature of every Clinical Congress.

HOSPITAL CONFERENCE

Dr. W. Edward Gallie, President of the College, will address the opening session of the twenty-fifth annual Hospital Standardization Conference on Monday morning at 10:00 o'clock in the Grand Ballroom, and will preside at the meeting, which will be attended by surgeons and hospital representatives. Following Dr. Gallie's résumé of the Hospital Standardization program of the College, including plans for the future, there will be reports on the progress of the 1946 Hospital Standardization survey, final results of which, including the Approved Lists of hospitals, cancer clinics, and approved hospitals for graduate training in surgery, will be announced at the end of the year, instead of at the Clinical Congress as was the practice in former years.

The remainder of the Monday morning session will be devoted to talks by medical and hospital authorities on advances in medicine and surgery as they affect the postwar hospital, with particular consideration of their relation to the Hospital Standardization program.

The afternoon conference on Monday will be concentrated upon current problems and the outlook for the future in nursing service. The Tuesday morning conference will have as its main subject personnel management, and the Tuesday afternoon session will be centered around personnel problems in specific fields, such as dietary, laboratory, medical records, medical social service, x-ray, physical and occupational therapy, and other professional and semi-professional services. The discussions will emphasize the importance of scientific management techniques in assuring high quality service from personnel.

The Wednesday morning conference will have as its main theme the importance of assuring high standards of care to the community and all patients through providing in the general hospital facilities for all types of illness. The subject will be discussed from the standpoint of psychiatric, tuberculous, cancer, chronically ill, and convalescent patients. The Wednesday afternoon conference will center mainly around discussion of ways of improving co-operation between general hospitals and special hospitals in the care of all types of special patients.

The Thursday morning conference will revolve around the physical plant of the hospital and the importance of improvement and modernization in raising standards of care of the patient. New mechanical and technical developments will be presented. The afternoon conference will be devoted to the hospital as the health center of the community, with consideration of the effects of this concept upon hospital public relations and upon the progress of preventive medicine and public health.

An evening conference will be held at 7:00 o'clock on Tuesday. This will be devoted to discussion of the responsibilities of trustees, and members of governing boards will be especially invited to attend and to participate. A conference will be held on Wednesday evening also. This will be a round table conference on responsibilities of hospital administrators, and the preparation which is necessary to enable them to cope successfully with the wide range of administrative problems. The discussion of these problems will interest all hospital personnel.

CLINICAL CONGRESS PROGRAM IN BRIEF

Monday

10:00 General Assembly for Surgeons and Hospital Representatives, Grand Ballroom
1:30–3:00 Panel Discussion, Empire Room
2:00 Clinics, Demonstrations and Clinical Group Conferences, Selected local hospitals
2:00 Hospital Conference, Sert Room
1:00 Surgical Film Exhibition (General), Grand Ballroom
3:30–5:00 Panel Discussion, Empire Room
8:00 Presidential Meeting, Grand Ballroom

Tuesday

9:00 Clinics, Demonstrations and Clinical Group Conferences, Selected local hospitals
10:00 Hospital Conference, Sert Room
9:30 Surgical Film Exhibition (Eye, Ear, Nose and Throat), Jensen Suite
9:30–12:00 Forum on Fundamental Surgical Problems, Grand Ballroom
10:00 Surgical Film Exhibition (General), Empire Room
11:00 Panel Discussions
Ophthalmology
Otorhinolaryngology
1:30–3:00 Panel Discussion, Grand Ballroom
2:00 Clinics, Demonstrations and Clinical Group Conferences, Selected local hospitals
2:00 Hospital Conference, Sert Room
2:00 Symposium on Fractures and Other Traumas
2:00 Surgical Film Exhibition (General), Empire Room
3:30–5:00 Panel Discussion, Grand Ballroom
7:00 Surgical Film Exhibition (Eye, Ear, Nose and Throat), Empire Room
7:00 Hospital Conference—Trustees, Sert Room
8:00 Scientific Session, General Surgery, Grand Ballroom
8:00 Scientific Session, Ophthalmology, Jensen Suite
8:00 Scientific Session, Otorhinolaryngology, Le Perroquet Suite

Wednesday

8:00 Meeting of Cancer Committee, Carpenter Foyer and Dining Room
9:00 Clinics, Demonstrations and Clinical Group Conferences, Selected Local Hospitals
9:30 Hospital Conference, Sert Room
9:30 Surgical Film Exhibition (Eye, Ear, Nose and Throat), Empire Room
9:30–12:00 Forum on Fundamental Surgical Problems, Grand Ballroom
10:00 Surgical Film Exhibition (General)
State and Provincial Committees:
9:30 Judiciary Committees
10:00 Executive Committees
10:30 Credentials Committees and Committees on Applicants
(Empire Room)
11:00 Panel Discussions
Ophthalmology
Otorhinolaryngology
12:00 Meeting of Board of Governors, Jensen Suite
1:30–3:00 Panel Discussion, Grand Ballroom
2:00 Clinics, Demonstrations and Clinical Group Conferences, Selected local hospitals
2:00 Symposium on Cancer, Empire Room
2:00 Surgical Film Exhibition (General)
2:30 Hospital Conference, Sert Room
3:30–5:00 Panel Discussion, Grand Ballroom
6:00 Vandyck Reunion Dinner, Le Perroquet Suite
7:00 Surgical Film Exhibition (Eye, Ear, Nose and Throat), Empire Room
7:30 Hospital Conference, Sert Room
8:00 Scientific Session, General Surgery, Grand Ballroom
8:00 Scientific Session (Eye, Ear, Nose and Throat), Empire Room

Thursday

9:00 Clinics, Demonstrations and Clinical Group Conferences, Selected local hospitals
9:30 Hospital Conference, Sert Room
9:30 Surgical Film Exhibition (Eye, Ear, Nose and Throat), Empire Room
9:30–12:00 Forum on Fundamental Surgical Problems, Grand Ballroom
10:00 Surgical Film Exhibition (General), Empire Room
11:00 Panel Discussions
Ophthalmology
Otorhinolaryngology
1:30 Adjourned Meeting, Governors, Grand Ballroom
1:45 Annual Meeting, Fellows, Grand Ballroom
2:00 Clinics, Demonstrations and Clinical Group Conferences, Selected local hospitals
2:00 Hospital Conference, Sert Room
3:00 Panel Discussion—Graduate Training in Surgery, Jensen Suite
3:30–5:00 Panel Discussion, Grand Ballroom
3:30 Surgical Film Exhibition (General), Empire Room
3:30 National and Regional Fracture Committees, Le Perroquet Suite
4:00 Committee on the Library, Room 4-J
7:00 Surgical Film Exhibition (Eye, Ear, Nose and Throat), Empire Room
8:00 Scientific Session, General Surgery, Grand Ballroom
8:00 Scientific Session, Ophthalmology, Empire Room
8:00 Scientific Session, Otorhinolaryngology, Sert Room

Friday

9:00 Clinics, Demonstrations and Clinical Group Conferences, Selected local hospitals
9:30 Surgical Film Exhibition (Eye, Ear, Nose and Throat), Empire Room
9:30–12:00 Forum on Fundamental Surgical Problems, Grand Ballroom
10:00 Surgical Film Exhibition (General), Empire Room
1:30–3:00 Panel Discussion, Surgery of the Stomach.
2:00 Clinics, Demonstrations and Clinical Group Conferences, Selected local hospitals
2:00–4:00 Panel Discussions
Obstetrics, Sert Room
Plastic Surgery, 4-U Blue Room
Neurological Surgery, Assembly Room M-N
Thoracic Surgery, Jensen Suite
Urology, Le Perroquet Suite
Orthopedic Surgery, Carpenter Foyer and Dining Room
2:00 Surgical Film Exhibition (General), Empire Room
3:30–5:00 Panel Discussion, Surgery of the Vascular System.
8:00 Convocation, Grand Ballroom

ANNUAL MEETING OF FELLOWS

The annual meeting of the Governors and Fellows will be held on Thursday afternoon at 1:45. There will be election of officers and governors. The annual meeting affords the Fellows of the College an opportunity to hear reports of officials on the work of the organization, and to learn how it has not only raised the professional and ethical standards of surgery, but has also promoted good hospitalization and general improvement in the practice of medicine in the United States and Canada. Each Fellow has a personal part in this work and may extend the influence of the College materially in his local community. Hospital Standardization alone offers him unlimited opportunity to provide better medical care for his patients in the hospital in which he works through continuous progress in applying the principles of the Minimum Standard which insure the best care of the patient.

Every Fellow will want to attend this important meeting, at which reports will be presented on financial affairs; Hospital Standardization; Graduate Training in Surgery; Medical Motion Pictures; Public Relations; Library and Literary Research; the work of the state and provincial credentials committees, committees on applicants, and the Committee on History Reviews; Sectional Meetings, 1946; and the Department of Clinical Research, including cancer clinics, medical service in industry, the Committee on Cancer, and the Committee on Fractures and other Traumas. Dr. Abell will report on administration of the College, staff changes, and retirements, and Dr. Arthur W. Allen, Vice-Chairman of the Board of Regents will discuss "Fellowships, Obligations and Opportunities."

STATE AND PROVINCIAL EXECUTIVE, CREDENTIALS AND JUDICIARY COMMITTEES

On Wednesday morning from 9:00 to 12:00 o'clock the State and Provincial Executive, Credentials, and Judiciary committees will meet to discuss their respective activities.

MEDICAL MOTION PICTURES

The latest available films showing surgical procedures and related subjects will be shown in the medical motion picture exhibits which will be held daily. These are a much appreciated feature of the Clinical Congress. Despite the decrease in production of such films during the war period, a surprising number of new pictures on varied subjects are being received by the American College of Surgeons for review.

The schedule will be so arranged as not to conflict with either the clinical program at the hospitals or the scientific sessions. Both sound and silent, standard and color films will be shown, all of which have been approved by the Committee on Medical Motion Pictures.

TECHNICAL AND SCIENTIFIC EXHIBITION

The technical exhibit, together with the registration and clinic ticket bureaus, will be located in the Basildon Room, Jade Room, and Astor Gallery, all on the third floor of the hotel. Leading manufacturers of surgical instruments, x-ray apparatus, sterilizers, operating room lights, ligatures, dressings, hospital apparatus and supplies of all kinds, pharmaceuticals, and publishers of medical books will be represented in the exhibition. The technical exhibits will demonstrate many of the newer features learned from our experience in the war.

EVENING SCIENTIFIC SESSIONS

GENERAL SURGERY

Tuesday

Care of the Patient before and after Operation

Fluid and Electrolyte Balance. CARL MOYER, Eloise, Michigan.
Nutritional Preparation of the Substandard Risk Patient. RICHARD L. VARCO, Minneapolis.
Use of Blood and Blood Substitutes. JOHN D. STEWART, Buffalo.
Blood Transfusion Problems, with Special Reference to the Rh Factor. ALEXANDER S. WIENER, Brooklyn.
Safe Conduct of the Patient Through Operation. OWEN H. WANGENSTEEN, Minneapolis.

Wednesday

Venous Thrombosis and Prevention of Pulmonary Embolism

Interruption of Deep Veins. ARTHUR W. ALLEN, Boston.
Sympathetic Nerve Block. ALTON OCHSNER, New Orleans.
Anticoagulants. D. W. GORDON MURRAY, Toronto.

Fracture Oration

Modern Methods in the Treatment of Fractures. EDWIN W. RYERSON, Chicago.

Thursday

Antibiotic and Chemotherapeutic Agents in Surgery

Pathologic Physiology of Surgical Infections. JOHN S. LOCKWOOD, New Haven.
Penicillin—Its Use in Surgery and Influence on Earlier Types of Chemotherapy. WILLIAM A. ALTEMEIER, Cincinnati.
Streptomycin. HORTON C. HINSHAW, Rochester.
Correlation of the Use of Antibiotic and Chemotherapeutic Agents with General Principles of Surgery. CHAMP LYONS, New Orleans.

OPHTHALMOLOGY

Tuesday

Orbital Reconstruction Including Prosthesis

Acrylic Prosthesis.
Basket Implant. NORMAN L. CUTLER, Wilmington.
Orbital Measurements.
Reconstruction of Upper Lid and Orbital Floor Defects. Major ARTHUR E. SHERMAN, Springfield, Missouri.

Thursday

Surgical Applied Anatomy in Eye Surgery. MEYER WIENER, Coronado.
Treatment of Glaucoma with Di-Isopropyl Flurophosphate. PHILLIP ROBB MCDONALD, Philadelphia.
Visual Disturbances Associated with Head Injuries.

OTORHINOLARYNGOLOGY

Tuesday

Treatment of Deafness

The Suppurating Ear.
The Fenestration Operation. GEORGE E. SHAMBAUGH, JR., Chicago
Use of Radium for Conductive Deafness. JOHN E. BORDLEY, Baltimore.

Thursday

Surgery of the Nasal Accessory Sinuses

Indications for Surgery in the Light of the Use of Antibiotics.
Intranasal Surgery. JOHN J. SHEA, Memphis.
External Operations.

SYMPOSIUM ON PLASTIC SURGERY OF THE HEAD AND NECK

Wednesday 8 p.m.

Cancellous Bone Grafts to the Jaw.
Repair of Nasal Defects with Free Composite Grafts of Skin and Cartilage from the Ear. JAMES B. BROWN, St. Louis.
Studies in the Anatomy and the Repair of Cleft Palate.
Preservation of Function Following Resections of Jaw Tumors.

PANEL DISCUSSIONS

GENERAL SURGERY

Monday, 1:30–3:00 p.m.

Rehabilitation of the Surgical Patient and Early Ambulation. Leader, HOWARD RUSK, New York.

Monday, 3:30–5:00 p.m.

The Treatment of Cancer of the Large Bowel. Leader, FREDERICK A. COLLER, Ann Arbor.

Tuesday, 1:30–3:00 p.m.

Thiouracil in Thyroid Disease. Leader, FRANK LAHEY, Boston.

Tuesday, 3:30–5:00 p.m.

Recent Trends in the Management of Carcinoma of the Cervix. Leader, JOE VINCENT MEIGS, Boston.

Wednesday, 1:30–3:00 p.m.

Anesthesia. Leader, HENRY K. BEECHER, Boston.

Wednesday, 3:30–5:00 p.m.

Protein Metabolism in the Surgical Patient. Leader, ROBERT ELMAN, St. Louis.

Thursday, 3:30–5:00 p.m.

Spinal Cord Injuries. Leader, HOWARD C. NAFFZIGER, San Francisco.

Friday, 1:30–3:00 p.m.

Surgery of the Stomach. Leader, ROSCOE R. GRAHAM, Toronto.

Friday, 3:30–5:00 p.m.

Surgery of the Vascular System. Leader, DANIEL C. ELKIN, Atlanta, Georgia.

SURGICAL SPECIALTIES

Friday 2:00—5:00

Orthopedic Surgery Panel. (Subject to be announced.) Leader, CARL E. BADGLEY, Ann Arbor.
Urology Panel. (Subject to be announced.) Leader, HERMAN L. KRETSCHMER, Chicago.
Plastic Surgery Panel. (Subject to be announced.) Leader, ROBERT H. IVY, Philadelphia.
Neurological Surgery Panel. (Subject to be announced.) Leader, FRANCIS C. GRANT, Philadelphia.
Obstetrics Panel. (Subject to be announced.) Leader, FREDERICK C. IRVING, Boston.
Thoracic Surgery Panel. (Subject to be announced.) Leader, RICHARD H. SWEET, Boston.

OPHTHALMOLOGY

Panel discussions in the field of Ophthalmology are planned for Tuesday, Wednesday, and Thursday mornings at 11:00 o'clock, on the following subjects: Retinal Detachment; Glaucoma; Keratoplasty.

OTORHINOLARYNGOLOGY

Panel discussions on subjects in the field of Otorhinolaryngology are planned for Tuesday, Wednesday and Thursday mornings at 11:00 o'clock on the following subjects, respectively: The Treatment of Ménière's Syndrome; Osteomyelitis of the Skull; Rehabilitation of War Deafness.

SYMPOSIUM ON FRACTURES AND OTHER TRAUMAS

At 2:00 o'clock on Tuesday afternoon there will be held a symposium on Fractures and Other Traumas under the direction of Robert H. Kennedy of New York. This symposium will be devoted to a number of brief presentations by specialists in various fields concerning new or improved methods adopted during the war which are applicable to injury in civilian surgery. The aim is to give a rapid survey of worthwhile gains made during the war which may prove of lasting value in the field of trauma.

Among the fifteen or twenty short papers which are planned are the following:

Bone Grafts. GEORGE K. CARPENTER, Nashville.
Spinal Cord Injuries. DAVID H. POER, Atlanta.
Injuries to the Rectum. EDMUND J. CROCE, Worcester.
Transportation of Fractures. WILLIAM J. STEWART, Columbia.
Replacement of Skin Defects. JAMES B. BROWN, St. Louis.
Fractures of the Carpal Scaphoid. MATHER CLEVELAND, New York.
Amputations. RUFUS H. ALDREDGE, New Orleans.
Training of Amputees. HENRY H. KESSLER, Newark.
Rehabilitation. Col. A. WILLIAM REGGIO, Washington.
Compound Fractures Treated with Penicillin and Delayed Primary Closure. OSCAR P. HAMPTON, Jr., St. Louis.

SYMPOSIUM ON CANCER

A symposium on cancer is planned for Wednesday afternoon. The Cancer Committee of the College has done outstanding work in furthering the development of cancer clinics in hospitals and providing for the registration of cured cases of malignant disease in the cancer archives. The chairman of this committee will give a brief review of these activities of the College in opening the symposium.

Other subjects of practical interest are under consideration for inclusion in the program, which is being planned to interest surgeons, pathologists, and radiologists.

PRELIMINARY CLINICAL PROGRAM—NEW YORK

BELLEVUE HOSPITAL

Monday

Anesthesia—2.
E. A. Rovenstine and staff. Dry clinic: Management of anesthesia for intrapleural surgery. Nerve block demonstrations: glosso-pharyngeal block for diagnosis; sympathetic block for phantom pain; paravertebral block for fracture of ribs; suprascapular block for shoulder pain; discussion of drugs and methods. Exhibit: A male moulage is utilized to demonstrate the landmarks for various nerve blocking procedures.
Neurosurgery—2.
Joseph King, Wilfred Wingebach, Lawrence Pool, Herbert Reikert. Operative clinics.
Ophthalmology (time to be announced).
Daniel B. Kirby. Cataract surgery; ptosis and strabismus surgery.
Arno E. Town. Cornea.
James Houlahan. Glaucoma.
Loren P. Guy. Lacrimal passages.
Radiation therapy—2.
Ira I. Kaplan, Rieva Rosh, Sidney Rubenfeld. Dry clinic: Head and neck cases.
Urology—2.
Robert Hotchkiss and Howard Jeck; John W. Draper and Joseph Reikert; Gustav Humphries and George Slaughter; Dean Makowski.

Tuesday

General surgery—Operative clinics—8.
John H. Mulholland, Thomas J. Galvin, Victor Carabba, Samuel Standard, Irwin E. Siris, John A. Lawler, H. M. Wertheim.
Dry clinic—10.
Samuel Standard. Results of anterior resection for carcinoma of rectum.
John A. Lawler and Elaine P. Ralli. Postoperative thyroid results.
Henry Doubilet. Pancreatic enzymes.
Co Tui. Protein metabolism.
Wallace B. Murphy. Management of static ulcers.
Victor Carabba. Results of repair of body defects by free fascial grafts.
Walter B. Crandell. Vagus nerve resection for ulcer of stomach and duodenum.
John Hall. Acute arteristis of the appendix.
Sigmund Wilens. Tissue reaction to talc.
Fractures and other traumas—round table discussion—2. Fracture dislocation of the surgical neck of the humerus. Fractures of the neck of the humerus. Fracture dislocations of the elbow. Plateau fracture of tibial condyles. Fracture dislocations of the ankle. Carnes Weeks, Herbert J. Riekert, Irwin E. Siris, Kenneth M. Lewis.
Obstetrics and gynecology: Operative clinic—8; Dry clinic—10.
Mortimer D. Speiser. Management of unengaged vertex in multiparae at full cervical dilatation.
Irving Perlmutter. Indications for therapeutic abortion at Bellevue Hospital, 1935–1945.
Herbert B. Guyer. Preclinical cancer of the cervix—case report of 7 year history.
Equinn W. Munnell. Management of the delivery of the second of twins.
Louise M. Dantuano. Staphlococcus bacteriemia—three cases.
Physical medicine—2.
George G. Deaver. Demonstration and motion picture: Rehabilitation of spinal cord injuries. Discussion: Evaluation of extent of disability; methods of treatment for ambulation and self-care.
Radiation therapy—2.
Ira I. Kaplan, Rieva Rosh, Sidney Rubenfeld. Dry clinic: Breast and miscellaneous cases.

Wednesday

General surgery: Operative clinics—8. Dry clinic—10.
Frank B. Berry and associates
Philip C. Potter, Carnes Weeks, Robert Wylie, Dwight B. Fishwick, James Bagg, Wallace W. Sheridan, Walter W. Fischer, Robert B. Hyatt, William Heroy, Aaron Himmelstein.
Chest service: Operative clinic—8. Dry clinic—2.
Frank B. Berry and associates
Herbert C. Maier, Adrian Lambert, Jr., Laurence Miscall, Charles W. Lester, Walter B. Crandell.

Thursday

General surgery: Operative clinics—8. Dry clinic—10.
Arthur S. McQuillan. Thyroid disease.
J. William Hinton. Stomach.
Lester Breidenbach. Colon.
Edward V. Denneen. Gall bladder.
L. Breidenbach. End-results: stricture of rectum from lymphogranuloma venereum.
Children's surgery: Operative clinics—2. Dry clinic—3:30.
Philip Allen. Hernia.
Charles Lester. Chest Surgery.
Dental surgery: Dry clinic—3:30.
Leo Winter.
Fractures and other traumas—8.
Kenneth M. Lewis. Fracture of femur (neck).
Orthopedic surgery: Operative clinics—2. Dry clinic—3:30.
John McCauley and Arthur Krida.

Friday

General surgery: Operative clinics—8:30.
Guilford S. Dudley. Abdominoperineal resection for carcinoma of the rectosigmoid.
Russell H. Patterson. Vascular operative clinic.
Cranston Holman. Subtotal gastrectomy for peptic ulcer.
Ernest W. Lampe. Thoracolumbar sympathectomy.
Dry clinic—10:30.
Laurence Miscall. Unusual cardiopulmonary wounds. Report on the diagnosis and treatment.
David Lyall and John Luomanen. Richter's hernia—a report on 17 cases.
John Luomanen and Prudden. Internal fixation of fractures of the neck of the femur.
Russell H. Patterson. The rôle of venogram in vascular surgery with a report of some unusual cases.
Samuel W. Moore. Peptic ulcer perforation—a report on a series.
Herbert Reikert. Gastrojejunocolic fistula—a report of 7 cases.
Otorhinolaryngology: Operative clinic—2. Dry clinic—3:30.

JOSEPH D. KELLY and staff. Laryngeal surgery. Presentation of cases.

Roentgenology Exhibition

Roentgen illustration of inflammatory and neoplastic lesions. LEWIS J. FRIEDMAN. Tuesday through Friday —2–4 P.M.

BETH ISRAEL HOSPITAL

Tuesday

ARTHUR J. BARSKY—9. Plastic surgery: Operations.
SEYMOUR F. WILHELM and staff—2. Urology: Operations.

Wednesday

SIDNEY W. GROSSE and staff—9. Neurosurgery: Operations.
HENRY C. FALK and staff—9. Gynecology: Operative and dry clinic.
PERCY KLINGENSTEIN and staff—2. Surgery: Operative and dry clinic.
EDWIN G. LANGROCK and SAMUEL J. SCADRON and staffs: 2. Operative obstetrics.

Thursday

Gynecology: Operative and dry clinic—9.
ARTHUR S. W. TOUROFF—2. Chest surgery.

Friday

IRVIN BALENSWEIG and staff—9. Orthopedics: Operative and dry clinic.
SOLOMON G. HERSHEY and staff—9. Anesthesiology.
EDWIN G. LANGROCK and SAMUEL J. SCADRON—2. Tumor clinic; obstetrical clinic.
Pantopaque myelography in diagnosis of herniated intervertebral disc.

FLOWER AND FIFTH AVENUE HOSPITALS

Monday

Neurosurgery—3.
ISADORE MAX TARLOV. Technique of nerve suturing and grafting by plasma clot.
THOMAS IRVING HOEN. End-results.

Tuesday

Breast Surgery—9.
HERBERT CLIFTON CHASE. Carcinoma of the breast; demonstrations and end-results.
Tumor clinic—11.
GEORGE THOMAS PACK, GEORGE HIGGINS and FRANK JAMES BORRELLI.
Thoracic surgery—2.
SAMUEL ALCOTT THOMPSON and staff. The results of surgical treatment of anginal disease.
Obstetrics—2.
HORACE ERNEST AYERS, L. S. LOUIZEAUX and staff. Demonstration on the Ayers mannequin; moving picture with sound and color.
Orthopedics and fractures—2.
MILTON JOHN WILSON, ALAN CANTWELL and staff. Fracture treatment with motion pictures and end-results demonstrations.

Wednesday

JEROME GEORGE KAUFMAN, C. HENRY MERSHEIMER, and THOMAS H. MCGAVACK—9. Surgery of the thyroid and the use of Thyurea preparations.
LANGE and JOHN S. O. HERRLIN, JR.—11. The test of viability of the bowel by the fluorescein method.
JOHN FOLKNER FORD and DAVID MCCULLAGH MAYER—2. Dry clinic showing the methods employed in reconstruction of the external ear.
Ophthalmology—2.
JOHN MILTON MCLEAN. Demonstration of the Barroquer phaco-erysis operation for cataract.
CHARLES AARON TURTZ and CARL CONRAD SALZMAN. Squint operation.

Thursday

JOSEPH H. FORBES, JOHN CLIFFORD HAYNER and ALAN GRAHAM—9. Hernia; a new operation for hernia; end-results; discussion of wound healing.
SAMUEL GLASSER, ADLER, and FRANCIS ANTHONY SILEO——10:30. Femoral vein ligation for phlebothrombosis; femoral vein ligation for chronic thrombophlebitis; femoral vein ligation for arteriosclerosis, thrombectomy, and embolectomy—indications and end-results.
Plastic surgery—2.
CLARENCE RALPH STRAATSMA. Plastic surgery in civil practice; operative and dry clinics.
Otolaryngology—2.
J. A. W. HETRICK and ARTHUR J. HERSIG. End-results of tonsillectomy.

Friday

JOHN S. HERRLIN—9. Accidents to the common duct. Indications for common duct surgery and methods for reconstruction.
FRANCIS DAVIS SPEER—10. Water balance.
JOHN S. HERRLIN—10. Surgical aspects of water balance.
CHARLES A. HALBERSTAM and MICHAEL GEORGE MULINOS—11. A new amino acid preparation; its clinical use.

GOLDWATER MEMORIAL HOSPITAL
THE THIRD SURGICAL (N.Y.U.) DIVISION

Monday through Wednesday (time to be announced)

WILLIAM H. BARBER, HAROLD B. KEYES and staff. Chronic ulcerative colitis.
WILLIAM H. WALKER and staff. Non-union and contractures.
GEORGE W. SLAUGHTER and staff. Genitourinary problems in the aged.
MARGARET STANLEY-BROWN, NED SHNAYERSON and staff. Peripheral vascular disease.

HARLEM HOSPITAL

Monday

Burns and plastic surgery symposium—2.
JOSEPH A. TAMERIN. Resurfacing of extensive burns. Clinical cases and lantern slides.
ARTHUR L. GARNES. The problem of shock in burns.
Gastric and biliary surgery symposium—3:15.
AUBRE D. MAYNARD. Problems in biliary surgery.
JOHN P. BRUCKNER. Gastric surgery.
FRANCIS X. TIMONEY. Use of sulfonamides in peptic ulcer.

Tuesday

Trauma symposium—9.
RALPH H. YOUNG. Ward rounds—male building, followed by discussion of basic principles of traumatic surgery and nontraumatic surgery.
DAVID H. SMITH. Head injuries: Presentation of cases.
AARON PRIGOT. Injuries of liver, traumatic rupture, stab wounds and functional tests.
ROBERT S. WILKINSON. Stab and gunshot wounds of abdomen.
MYRA A. LOGAN. Stab wounds of the heart.
Roentgenology in surgery—2.
WILLIAM SNOW. Duodenal loop triad.
LEON GINZBURG. Intestinal obstruction and regional ileitis.

Genitourinary conference—3:30.
NORMAN F. LASKEY and staff. Kidney and bladder trauma with presentation of cases; urethral stricture; renal calculi.

Wednesday

FRANCIS X. TIMONEY. Ward rounds, Woman's building. Traumatic and nontraumatic cases.
LOUIS T. WRIGHT and JOSEPH LEVY. The problem of rectal stricture; presentation of cases.
BENJAMIN N. BERG. Pancreatic disease.
Gynecological symposium—2.
HENRY C. FALK and staff. Operative clinic in gynecological surgery; dry clinic: pelvic inflammatory disease.

Thursday

Fracture and orthopedic symposium—9.
ARTHUR C. LOGAN, WILLIAM A. FREEMAN, ANTHONY T. PRIVITERA. Fractures of the femur, with demonstrations and correlated operations.
JOSEPH G. WISHNER and staff. Slipped epiphysis with clinical demonstration.
SAMUEL SHENKMAN. Treatment of cervical spine with fracture.
LOUIS T. WRIGHT. Traumatic ward rounds.
Obstetrical symposium—2.
FREDERICK A. KASSEBOHN. Obstetrical emergencies and their treatment.
ABRAHAM C. POSNER. Recent advances in the toxemia of pregnancies.
JULIUS KURZROCK. The technique of Barton's forceps.
M. SCHREIBER. Analgesia in obstetrics.
NORMAN P. PLESHETTE. Tumors complicating pregnancy and labor.
WILLIAM SNOW. Soft tissue diagnosis by x-ray in obstetrics.

Friday

Thyroid disease—9.
ALEXANDER ALTSCHUL. Medical aspects of thyroid disease.
JESSE J. GREENE. Surgical aspects of thyroid disease.
Symposium on chest diseases—9:30.
JAMES C. WHITAKER. Injuries of the chest.
FARROW R. ALLEN. Thoracoplasty.
COSTABILE DI LORENZO. Surgical diseases of the chest.
DOMINICK F. ZETENA—10:10. Pediatric surgery.
ANGELO A. ZINGARO—11. Diverticulitis.
JAMES L. WILSON and ROBERT L. NACH—11:30. Amputation and the use of refrigeration anesthesia.
ANTHONY T. PRIVITERA—9. Traumatic ward rounds.
LOUIS T. WRIGHT—2. Mortality conference.
SOLOMON WEINTRAUB—2. Surgical pathology demonstrations.
The staff—2. Unusual cases.

HARLEM EYE AND EAR HOSPITAL

Wednesday

Otorhinolaryngology—1. Operations—Tonsillectomies with Meding tonsil enucleator.
Ophthalmology—2. Cataract extraction—Caldwell Luc.
Otorhinolaryngology—3. Demonstration of ear, nose and throat cases.

HOSPITAL FOR JOINT DISEASES

Monday

LEO MAYER and staff—8. Operative clinic. Tendon transplantation on the foot for traumatic division of flexor tendons; tendon transplantation in the hand for traumatic division of flexor tendons: fusion of the hip by new approach.
SAMUEL KLEINBERG and staff—2. Dry clinic. Demonstration of cases illustrating results of tendon transplants in the hand and foot and fusion operations on the hip: epiphysiolisthesis—results of drilling in early cases; chronic osteomyelitis—results of operation combined with use of penicillin; flat feet—indications for and results of Miller operation; club feet—manipulative and operative treatment; Perthes' disease.

Tuesday

SAMUEL S. KLEINBERG and staff—8. Operative clinic. Spine fusion for chronic low back pain; herniation of intervertebral disc (A. KAPLAN); arthrotomy for internal derangement of the knee; epiphysiolisthesis—drilling; contracted toes—plastic operation; congenital dislocation of the hip—open reduction.

Wednesday

ISADORE I. ZADEK—8. Orthopedic cases—operations: treatment of ununited fracture of hip. Obturator nerve avulsion for painful hip.
HARRY GOLDMAN—8. Operative clinic: Hemorrhoids; fistula-in-ano; benign adenoma of rectum.
PAUL WILLIAM ASCHNER—1:30. Operations on kidney and bladder.
HARRY DAVID SONNENSCHEIN—2. Dry clinic orthopedic cases.

Thursday

HARRY DAVID SONNENSCHEIN and staff—8. Operations: arthrotomy of knee; lumbosacral fusion; transcervical plastic for radial nerve lesion.
HAROLD BERTRAM DAVIDSON and staff—1:30. Total hysterectomy for fibroids.

Friday

ABRAHAM JACOB BELLER and staff—8. Operations for carcinoma of stomach and colon.

HOSPITAL FOR SPECIAL SURGERY

Tuesday

PHILIP DUNCAN WILSON and staff—9. Childrens' orthopedic problems.
LEWIS CLARKE WAGNER—9. Severe spondylolisthesis—case presentation.
General surgical operations—8:30.

Wednesday

Children's orthopedic surgery—8:30.
CARL GOODWIN BURDICK and staff—9; 2. Hernia and special surgical problems.
Plastic surgery—9; 2.
YOLANDE E. H. HUBER and JOHN MARQUIS CONVERSE. Presentation of cases; motion pictures; plastic surgical operations.

Thursday

Surgery of bones and joints—9; 2.
PHILIP DUNCAN WILSON and staff. Adult orthopedic problems.
LEWIS CLARKE WAGNER. Wedge ostectomy of neck of femur for badly displaced upper femoral epiphysis.
L. RAMSAY STRAUB. Spine fusion with plate fixation.
PHILIP DUNCAN WILSON. Trochanteric cup arthroplasty.
THEODORE C. THOMPSON; LEWIS CLARKE WAGNER. Restoration of active knee motion.
ROBERT LEE PATTERSON, JR. Conservative treatment of fractures of the tibial plateau.
WILLIAM MORRIS COOPER. Aseptic necrosis of the head of the femur in adults.
PETER RIZZO. Bone graft with metallic fixation.
JOHN J. FLANAGAN. Massive tibial bone grafts.

Friday

Surgery of bones and joints—8:30.
RICHARD FREYBERG and staff. Arthritic problems.
Adult orthopedic surgery.
JOHN ROBERT COBB and staff—2. Scoliosis—idiopathic and paralytic.

LENOX HILL HOSPITAL

Monday

Obstetrics and gynecology—2.
ROBERT MCCREADY and MORTIMER ROGERS. Total hysterectomy by combined vaginal and abdominal routes.
Operative treatment of prolapse.
NATHAN BRISTOL VAN ETTEN. Total abdominal hysterectomy.
EDWARD P. KILROE. Vaginal hysterectomy.
CUNNINGHAM. Manchester-Fothergill operation.
BOYNTON. LeFort operation.
CARL GOLDMARK. Obstetrical analgesia—demerol versus barbiturates.

Tuesday

Surgery—9.
JOHN C. GERSTER, FRANCIS M. DONEHUE, HERBERT W. MEYER, ERICH FRICKE. Choice of operative procedures in lesions of colon and rectum.
Thoracic surgery—2.
C. EGGERS. Thoracic cases.
HERBERT CAILLE MAIER. Results of lobectomy and pneumonectomy in bronchiectasis, cystic disease of the lung and pulmonary abscess.
FISCHER. Surgical treatment of adenoma of bronchus.

Wednesday

Surgery—9.
OTTO CARL PICKHARDT. Treatment of hiatus hernia in older people by phrenemphraxis.
GEORGE ERNEST MUEHLECK. Acute pancreatitis.
HUBER. Symptomatology, diagnosis.
BLAKE FERGUSON DONALDSON. Operative procedure.
LEROY ALVIN WIRTHLIN. End-results.
Genitourinary surgery—2.
WILLIAM ROBERT DELZELL. Unusual calculi of urinary tract.
MAXIMILIAN MARK NEMSER. Polycystic kidney disease.
SLAUGHTER. Treatment of rupture of urinary bladder.
Ophthalmology—2.
CHRISTIAN FREDERICK REID. Transplantation of mucous membrane for eye burns and keratitis.
CHRISTIAN F. KETELS, JOHN HENRY BORN, RUDOLPH AEBLI, BERNARD FREAD, SAUER, ERNEST LUDWIG METZGER. Cataract surgery. Dry clinic postoperative: cases of retinal detachment.
Ophthalmology clinic—2.
RUDOLPH AEBLI and staff. Cataract operations; clinic on retinal detachments (demonstration of cases).

Thursday

Diagnostic-Bronchoscopic clinic—9.
ARTHUR JOSEPH CRACOVANER, HIRSCH. Bronchoscopy—indications and demonstration.
HENRY PAUL SCHUGT. Tuberculosis of larnyx; abscess of larnyx.
ERNEST ALFRED WEYMULLER. Aero-otitis and sinusitis—irradiation therapy.
J. E. JOHNSTONE KING. Neurosurgery.
Hematology and vascular surgery symposium—2.
CARL REICH. Heparin and dicumarl.
RICHARD WILLIAM KESSLER. Anatomy and operative procedures.
ECHLIN. Neurosurgery procedures.
MCWILLIAMS. Venography.
GEIGER. Rh factor and blood bank demonstration.

Friday

Surgery of the bones and joints—9.
WALTER ISAAC GALLAND. Osteotomy for oseteoarthritis of hip.
WALKER ELY SWIFT. Fracture—dislocation of ankle.
ALFRED FRANZ WIESENTHAL. Hallux valgus—management and end-results.
HENRY H. JORDAN. Roger Anderson pinning of fractures—end-results; hospital brace shop demonstration.
MADGE C. L. MCGUINESS. Discussion of general physical therapy procedures.
Plastic and faciomaxillary surgery—2.
FRED SQUIER DUNN. Cleft palate.
GUSTAVE AUFRICHT. Mammaplasty.

LYING-IN HOSPITAL

Monday

HENRICUS JOHANNES STANDER presiding—2. Presentation and discussion of interesting patients.

Tuesday

BYRON HEAZELTON GOFF and JAMES AITKEN HARRAR. Operative clinic: Vaginal plastic operations; obstetrical operations.
CHARLES MORDECAI MCLANE. X-ray pelvimetry.
JACOB THEODORE SHERMAN. The obstetrical pelvis from a clinical aspect.
FREDERICK WALL FINN. Evaluation of myomectomies.
JOHN T. COLE. Maternal obstetrical paralysis.

Wednesday

ANDREW ANTHONY MARCHETTI. Ectopic pregnancy.
CURTIS LESTER MENDELSON. Management of labor in cardiac disease.
JOHN T. COLE and BRUCE WILLIAMS. Clinical application of our knowledge of Rh factor; a report of two transfusion deaths due to Rh incompatibility.
ARTHUR VOSE GREELEY. Selective conservative management of endometriosis.

Thursday

HENRICUS JOHANNES STANDER—9. Gynecology operative clinic.
JAMES AITKEN HARRAR—9. Obstetrical operations.
CHARLES MORDECAI MCLANE—2. Sterility.
ROBERT GORDON DOUGLAS—2. Rupture of the uterus. Experiences in the Lying-In Hospital since 1890.
ROBERT GORDON DOUGLAS and R. LANDSMAN—2. Recent experience in the reduction of morbidity and puerperal infection in cesarean section.
WILLIAM GIVEN—2. Carcinoma of the cervix.

Friday

ANDREW ANTHONY MARCHETTI—9. Operative clinic.
HOWARD SHEILD MCCANDLISH—9. Obstetrical operations.
JOHN T. COLE and FORBES DELANEY. Transverse presentation.
ANDREW ANTHONY MARCHETTI—2:30. Clinical significance of vaginal smears.
CARL THEODORE JAVERT—2:30. Combined clinical procedure for anteroversion of retroverted uteri.
WILBUR DIXON—2:30. Management of face presentation.
FRANK SMITH—2:30. Missed abortion.

MANHATTAN EYE, EAR AND THROAT HOSPITAL
OPHTHALMOLOGY

Monday

G. BONNOCCOLTO—2. Slit lamp demonstration.
HERBERT MAURICE KATZIN—2. Demonstration corneal grafting technique on rabbits.

CHARLES HUGH BRANIGAN—3. Demonstration of cases of cataract surgery complicated by glaucoma.

Tuesday

WALTER GUERNSEY FREY—9. Surgical treatment of ptosis of lids; operation and end-results.
FRANK CONRAD KEIL—2. Surgical treatment of strabismus; operations and end-results.
FRANK CONRAD KEIL, JR. and MISS E. KNAUBER—2. Orthoptic clinic.

Wednesday

ARTHUR LINKSZ—9. Demonstration of aniseikonia.
MURRAY A. LAST—9:30. Plastic operations of eye.
FERDINAND L. P. KOCH—9:30. Demonstration glaucoma clinic; late results of glaucoma surgery.
HERBERT MAURICE KATZIN—10. Demonstration of corneal grafting technique on rabbits.
RICHARD TOWNLEY PATON and HERBERT MAURICE KATZIN—2. Demonstration of corneal grafting technique; corneal research laboratory.
DAVID HENRY WEBSTER—2. Cataract surgery.
ANDREW ANDERSON EGGSTON and A. B. PAUL—2. Allergy in ophthalmology.
JOSEPH LAVAL—3. Eye pathology; demonstration microscopic sections.

Thursday

G. BONNOCCOLOTO—9. Surgery; cataract extraction; corneal transplant; dacryocystorhinostomy.
FRANK CONRAD KEIL, JR.—10. Laboratory demonstration bacteriology of the eye.
RICHARD TOWNLEY PATON—2. Surgery; corneal graft.
MAURICE LENZ, CLARA LOWENHERZ OKRAINETZ—2. Radiation therapy in ophthalmology.
RICHARD TOWNLEY PATON—3. Demonstration of postoperative corneal graft cases.

Friday

HERBERT MAURICE KATZIN—10. Demonstration corneal grafting technique on rabbits.
FRANK CONRAD KEIL, JR., and MISS E. KNAUBER—2. Orthoptic clinic.

MANHATTAN EYE, EAR AND THROAT HOSPITAL OTOLARYNGOLOGY

Monday

WILLIAM RICHARD CASHION. Biplane flouroscopy and laminagraphy.

Tuesday

WILLIAM BREWSTER ALLAN and ALFRED SCHATTNER—9. Nasal plastic operation.
MISS WHITEHURST and MRS. NERENBERG—9:30. Demonstration; audiometry; selection and fitting of hearing aids; retraining residual hearing.
DAVID SYLVESTER CUNNING—10. Demonstration of postoperative cases of laryngectomy; esophageal voice training.
DANIEL SYLVESTER CUNNING—2. Operation: laryngectomy.
ANDREW ANDERSON EGGSTON and DANIEL SYLVESTER CUNNING—3. Clinicopathological demonstration; malignancy of the larnyx.

Wednesday

MARVIN FISHER JONES—9. Fenestration: operation and demonstration of postoperative cases.
ANTHONY NIGRO—10. Operation for polypoid sinusitis and demonstration of postoperative cases.
DANIEL SYLVESTER CUNNING—1:30. Bronchoscopic clinic; diagnosis and treatment.
ANDREW ANDERSON EGGSTON and A. B. PAUL—2. Allergy in otorhinolaryngology; demonstration of cases and testing technique.
W. H. TURNLEY and G. S. RYAN—2. Tonsil surgery.

Thursday

JOSEPH DOMINIC KELLY—10. Demonstration of results in cases of arytenoidectomy.
MAURICE LENZ and CLARA L. OKRAINETZ—2. Results of radiation in laryngeal tumors.
HENRY DAVID TATERKA—2. Demonstration of radiation of hypertrophied lymphoid tissue of nasopharynx.
MARVIN FISHER JONES—2. Operation: fenestration.
JOSEPH DOMINIC KELLY—2. Operation: arytenoidectomy.

Friday

MISS WHITEHURST and MRS. NERENBERG—9:30. Demonstration; audiometry, selection and fitting of hearing aids; retraining residual hearing.

MEMORIAL HOSPITAL

Monday

Operative Clinics—8.
FRANK E. ADAIR and staff. Breast tumors.
GEORGE E. BINKLEY and staff. Rectal tumors.
BRADLEY L. COLEY and staff. Bone tumors.
HAYES E. MARTIN and staff. Intraoral tumors.
GEORGE T. PACK and staff. Gastric tumors.
CORNELIUS P. RHOADS—2. Steroid patterns in neoplastic diseases.
HAYES E. MARTIN—2:30. Late results in cancer of mouth and pharynx.
WILLIAM L. WATSON—3. Intrathoracic tumors.
FRANK W. FOOTE, JR. and LEO MARINELLI—3:30. Radio iodine in diseases of the thyroid.
F. HOMBURGER—4:30. Protein and carbohydrate metabolism in cancer cases.

Tuesday

Operative clinics—8.
FRANK E. ADAIR and staff. Breast tumors.
HAYES E. MARTIN and staff. Intraoral tumors.
GEORGE T. PACK and staff. Gastric tumors.
HOWARD C. TAYLOR and staff. Gynecologic tumors.
WILLIAM L. WATSON and staff. Intrathoracic tumors.
HOWARD C. TAYLOR, JR.—2. End-results in cancer of cervix uteri.
FRANK WM. FOOTE, JR.—2:30. Early cancer of cervix uteri.
FRANK E. ADAIR—3:00. Hormone therapy in cases of advance mammary cancer.
FRED W. STEWART—3:30. Common errors in cancer diagnosis. Pathological aspects.
DAVIS KARNOFSKY and JOSEPH H. BURCHENAL—4:00. Use of nitrogen mustard in selected types of neoplastic disease.

Wednesday

Operative clinics—8.
FRANK E. ADAIR and staff. Breast tumors.
GEORGE E. BINKLEY and staff. Rectal tumors.
BRADLEY L. COLEY and staff. Bone tumors.
HAYES E. MARTIN and staff. Intraoral tumors.
HOWARD C. TAYLOR and staff. Gynecologic tumors.
LLOYD F. CRAVER—2. Late results in Hodgkin's disease.
GEORGE T. PACK—2:30. Transthoracic cardiectomy for gastric cancers.
GEORGE E. BINKLEY—3. Abdominoperineal resection.
DOUGLAS A. SUNDERLAND—3:30. Rectal lesions discovered in supposedly normal individuals in a cancer prevention clinic.

JULES C. ABELS—4:00. Postoperative management of cases of gastrointestinal malignancies.

Thursday

ROBERT S. SHERMAN—10. Round table roentgenographic conference.
HELEN Q. WOODARD—2. Effect of radiation on normal and neoplastic bone.
BRADLEY L. COLEY—2:30. Analysis of late results.
ARCHIE L. DEAN—3. Treatment after bladder operations.
GEORGE T. PACK—3:30. End-results in malignant melanoma.
SOPHIE SPITZ—4. Infantile neuronaevi—their confusion with malignant melanoma.

Friday

Operative clinics—8.
FRANK E. ADAIR and staff. Breast tumors.
HAYES E. MARTIN and staff. Intraoral tumors.
GEORGE T. PACK and staff. Mixed tumors.
HOWARD C. TAYLOR and staff. Gynecologic tumors.

MONTEFIORE HOSPITAL

Tuesday

SAMUEL SILBERT—2. Mid-leg amputations for gangrene in the diabetic.

Wednesday

SYLVAN LEWIS HAAS—9. Orthopedic surgery in arthritic and paralytic diseases.

Thursday

ISIDOR KROSS—2. Chest surgery—operative and dry clinic.

Friday

SAMUEL STANDARD—2. Physiological derangements in organic disease of the intestinal tract—end-results of surgery.
ARTHUR HARVEY GLICK—2. Errors in diagnosis in tumors of the large bowel.
DANIEL LASZLO—2. Conference on neoplastic diseases and demonstration.

MOUNT SINAI HOSPITAL

Monday

Gynecological clinic—8:30.
MORRIS AARON GOLDBERGER. Parametrial fixation operation (Fothergill).
MORRIS AARON GOLDBERGER. Result of parametrial fixation operations; postoperative follow-up (ten years).
JOSEPH ARNOLD GAINES. Conservative surgery for pelvic endometriosis.
EMANUEL KLEMPNER. The value of the 24 hour pregnancy test: A review of 1,000 cases.
ARTHUR MAXWELL DAVIDS. The clinical value of hysterography.
ROBERT ISADORE WALTER. The management of the menopause.
Neurological Surgery—2.
ABRAHAM KAPLAN. Long time follow-up in some neurosurgical cases (case presentations).
B. SCHLESINGER. Correlation of x-ray findings and clinical picture.
IRA COHEN. Dumbbell tumor of the spine.
S. GROSS. Cranioplasty, motion picture demonstration.

Tuesday

Surgery of the colon and rectum—8:30. Operative clinic.
JOHN HARRY GARLOCK. Anterior resection of the rectum; colectomy for ulcerative colitis.
JOHN HARRY GARLOCK, LEON GINZBURG, SAMUEL HAROLD KLEIN, AMEIL GLASS, GABRIEL PARKURST SELEY. Dry clinic and case presentations.
Orthopedic clinic—2.
ROBERT KORN LIPPMANN, ALBERT JACOB SCHEIN, EDGAR MILTON BICK. Operative and dry clinic.

Wednesday

Thoracic surgery—8:30.
ARTHUR SIGMUND TOUROFF. Operation for patent ductus arteriosus .
HAROLD NEUHOF. Intramuscular heparin (concentrated) therapy in the prevention of postoperative thrombosis and embolism.
Motion picture demonstration. Technique of operation for clotted hemothorax.
ARTHUR SIGMUND TOUROFF. Results of operative treatment of traumatic thoracic lesions in civilian practice.
ERNEST EMANUEL ARNHEIM. Operative results in pediatric surgery.
EDWARD E. JEMERIN. Results of surgical treatment of perforation of the esophagus.
IRVING ARTHUR SAROT. Results of conservative surgical treatment of chronic pulmonary abscess.
Clinic on ophthalmology—2.
ROBERT LAMBERT, HENRY MINSKY, JOSEPH LAVAL, DAVID WEXLER. The surgical treatment of glaucoma—operative clinic and dry clinic of operated cases.

Thursday

Surgery of the stomach and duodenum: Operative clinic—8:30.
RALPH COLP. Supradiaphragmatic vagotomy for duodenal ulcer; subtotal gastrectomy and infradiaphragmatic vagotomy for duodenal ulcer with stenosis.
Dry clinic:
FRANK HOLLANDER. Physiologic differences between gastric and duodenal ulcers.
LEONARD DRUCKERMAN. Palliative gastrectomy in selected cases of chronic gastric ulcer.
PERCY KLINGENSTEIN. Problems in the treatment of gastrojejunocolic fistula.
SIGMUND MAGE. Follow-up results in subtotal gastrectomy for gastric and duodenal ulcer.
STEPHEN ROSENAK. The advantages of jejunostomy for jejunal alimentation.
Ear, nose and throat clinic—2.
Inflammatory and neoplastic diseases of the frontal sinuses.
Carcinoma of the larynx and results, treatment by radiation therapy.
Operative and dry clinic.
RUDOLPH KRAMER, MORRIS BENDER, IRVING BRICE GOLDMAN, JOSEPH DRUSS, SAMUEL ROSEN and staff.

Friday

Genitourinary surgery—8:30. Operative clinic.
LEO EDELMAN. Kidney operation.
GORDON DAVID OPPENHEIMER. Carcinoma in a horseshoe kidney (dry clinic).
MOSES SWICK. Bladder neck obstruction in young individuals.
WILLIAM MENCHER. Perirenal insufflation.
H. EVANS LEITER. Enuresis due to ectopic ureter.

METROPOLITAN HOSPITAL

Monday

Neurosurgery—2.
I. W. OLJENICK.

Tuesday

EDWARD J. MCCABE, KENNETH C. PEACOCK and WILLIAM GREENWALD—9. Hemorrhage, perforation and the indications for surgery in peptic ulcer.

CHARLES A. HALBERSTAM, GEORGE HERLITZ and EUGENE FIERRO—11. Diagnosis and management of intestinal obstruction.

Urology—2.

ALFRED HUNT and LEONARD P. WERSHUB. Total perineal prostatectomy.

GEORGE R. NAGAMATSU. Plastic operations for ureteropelvic junction obstruction.

SPRAGUE CARLETON. General discussion.

Wednesday

HAROLD E. CLARK, WALTER GRAY CRUMP and WALTER L. MERSHEIMER—9. Colon surgery; Indications, methods and end-results.

H. B. SAFFORD and JOHN E. TRITSCH—10. Gynecology staff with presentation and consideration of problem cases.

Thoracic surgery—2.

SAMUEL A. THOMPSON and staff. Value of cavernostomy in tuberculosis.

Thursday

STEPHEN CHILIAN, LOUIS M. PALERMO and WILLIAM L. PRIMACOVE—9. Ecchinococcus cyst; diagnosis and management.

LOUIS R. KAUFMAN, LINN J. BOYD and CHRISMAN G. SCHERF—10. Surgery of hypertension; indications, technique and end-results.

JOHN F. FORD and DAVID M. MAYER—10. The problem in skin grafting for thrombophlebitis.

HARRY BAROWSKY—11. Round table discussion of abdominal diagnosis; gastroscopy.

LEONARD P. WERSHUB—11. Perinealoscopy.

JOHN HERRLIN and staff—11. Peritoneal tap and x-ray diagnosis.

MILTON JOHN WILSON and staff—2. Demonstration of end-results in spine and hip injuries.

Friday

SAMUEL THOMAS GLASSER, JOSEPH A. SILEO, and G. ADLER—9. Peripheral vascular diseases: end-results in refrigerating anesthesia; amputation; lumbar sympathectomy; multiple nerve section; lumbar ganglion block; intra-arterial penicillin.

SAFFORD and JOHN E. TRITSCH—10. Gynecology staff with presentation and consideration of problem cases.

JOHN F. FORD and DAVID M. MAYER—10. The problem in skin grafting for thrombophlebitis.

K. LANGE, LINN J. BOYD, DAVID O. WEINER, and ALBERT LESSER—10:30. Frostbite, its physiology and morphologic pathology and the prevention of subsequent gangrene.

NEW YORK EYE AND EAR INFIRMARY

OTOLARYNGOLOGY

Demonstrations and lectures daily by EARL FREDERICK LIMBACH, MAXWELL DONNELL RYAN, JAMES MORRISSET SMITH, JAMES SWIFT HANLEY, STUART LESSLEY CRAIG.

OPHTHALMOLOGY

Demonstrations and lectures daily by WENDELL LOCHEAD HUGHES, RAYMOND EMORY MEEK, CONRAD BERENS, BRITTAIN FORD PAYNE, WILLIS SACKETT KNIGHTON.

THE NEW YORK HOSPITAL

Monday

General surgery—2.

ERNEST WILLIAM LAMPE. Surgical anatomy demonstration: detailed anatomy of hand and wrist as involved in infection and trauma.

Otolaryngology: Operative clinic—2.

ARTHUR PALMER. Reconstructive surgery of the nose.

Tuesday

General surgery—9.

GEORGE JULIUS HEUER and staff. Operative and dry clinics.

GEORGE JULIUS HEUER. Follow-up study of treatment of peptic ulcer.

CRANSTON WILLIAM HOLMAN. Bleeding peptic ulcer.

WILLIAM COOPER. Diagnosis of stomach lesions.

EDGAR COOPER PERSON, Jr. Gastrojejunocolic fistula.

WILLIAM BARNES. Carcinoma of the head of the pancreas.

General surgery, dry clinic—2.

GEORGE JULIUS HEUER and staff.

WILLIAM DE WITT ANDRUS. Follow-up results. Aneurysm of large vessels.

WILLIAM COOPER. Follow-up results—carcinoma of breast, stomach, colon, rectum.

BRONSON SANDS RAY. Organization of follow-up clinic. Follow-up clinic staff. Demonstration.

ERNEST WILLIAM LAMPE. Surgical anatomy demonstration—anatomy of neck as regards radical neck dissection.

Genitourinary surgery—9.

ERNEST WILLIAM LAMPE. Surgical anatomy demonstration of male perineum.

Otolaryngology: operative clinic—2.

GERVAIS WARD MCAULIFFE. Surgery of the mastoid.

Urology (Cornell)—2.

VICTOR MARSHALL.

E. CRAIG COATS. Results of untreated bladder carcinoma.

VICTOR FRAY MARSHALL. Results of radiation treatment.

MARTIN SPATZ. Autopsy findings.

ARCHIE LEIGH DEAN, JR. Physical factors limiting radiation treatment.

JEREMIAH EDWIN DREW. Results in bladder resection.

ALLISTER MATHESON MCLELLAN. Results of fulguration and simple excision.

GUSTAVUS ALDRIDGE HUMPHREYS. Results of ureterocutaneous anastomosis.

DAVID H. MACFARLAND. Results of ureterointestinal anastomosis.

MORRIS SCHNITTMAN. Results of cystectomy.

VICTOR FRAY MARSHALL. Summary.

Wednesday

General surgery—2.

ERNEST WILLIAM LAMPE. Surgical anatomy demonstration—anatomy of breast as regards radical mastectomy.

Genitourinary surgery—2.

THOMAS JOSEPH KIRWIN. Operative and dry clinic: perineal prostatectomy.

ROSE HENRI ANDRÉ. Demonstration of spinal anesthesia.

ROY BIGGS HENLINE. Ureteropelvic obstruction.

GEORGE ADOLPH FIEDLER. Absorbable gauze in urologic surgery.

FRANCIS PATTON TWINEM. Case report.

ROBERT W. HUNT. True adrenal tumor of kidney.
CYRIL KLOCK CHURCH. Ureteral obstruction in relation to psychosis.
STANLEY LEON WANG. Treatment of bladder tuberculosis with a new instrument and method.
FRANCIS ANTHONY BENEVENTI. Glands in female urethra of the infant.
ALBERT VERGES-FLAQUE. Foreign body in ureter.
ALFONSO DAVALOS. Transplantation of ureters.
THOMAS F. CONROY. Case report..
ALBERTO GENTILE. Case report.
DENNIS ALOYSIUS KELLEHER. Gastroenterological symptoms in urology.
CECIL HAWES. Case report.
WILLIAM P. DIDUSCH. Demonstration of drawings of interesting urological cases and operations. Motion pictures of urological operations.

Neurology—2.
BRONSON SANDS RAY, HAROLD GEORGE WOLFF, JOSEPH HINSEY.
BRONSON SANDS RAY. Surgical treatment of cardiac pain.
CHARLES M. BERRY and JOSEPH HINSEY. Studies in regeneration of peripheral nerves.
CHARLES LAMAR NEILL, JR. Intracranial aneurysms.
DONALD SIMON and HAROLD GEORGE WOLFF. Studies of post-traumatic headache.
HERBERT PARSONS. Ruptured disks of the cervical region.
ARTHUR CONSOLE. Sympathetic innervation of the extremities.
BRONSON SANDS RAY. Results of sympathectomy for hypertension.
BRONSON SANDS RAY and staff. Neurosurgical case reports.

Thoracic surgery—9.
GEORGE JULIUS HEUER and staff. Operative and dry clinic.
GEORGE JULIUS HEUER. Constrictive pericarditis.
WILLIAM deWITT ANDRUS. Surgery of mediastinal tumors.
JOHN HAAG ECKEL. Follow-up studies of mediastinal tumors.
HENRY ARNOLD CROMWELL and WILLIAM ALEXANDER BARNES. Clinical value of sputum diagnosis.

Otolaryngology: Operative clinic—2.
SAMUEL F. KELLEY. Surgery of the paranasal sinuses.

Thursday

General surgery—2.
GEORGE JULIUS HEUER. History of operative procedure for acute cholecystitis.
NATHAN CHANDLER FOOT. Pathology of acute cholecystitis.
FRANK GLENN. Surgical treatment of acute cholecystitis.
SAMUEL WILSON MOORE. Complications of acute cholecystitis.
ERNEST WILLIAM LAMPE. Surgical anatomy demonstration—anatomy involved in lumbar and thoracolumbar sympathectomy.

Plastic and faciomaxillary surgery—9.
HERBERT CONWAY. Operative and dry clinic; one stage push back operation for congenitally short palate; results following treatment of carcinoma of the head and neck: case presentations.
E. FREUD. Speech therapy in cases of cleft palate.
CHARLES NEWMAN. End-results following radical breast amputation with split skin grafting.
JAMES DINGWALL. Adult tissue extract in wound healing.

Otolaryngology: Dry clinic—2.
ARTHUR PALMER and associates. Case presentations and postoperative result—laryngofissure, carcinoma of antrum, mastoiditis with petrositis and meningitis, acute frontal sinusitis with meningitis.

Ophthalmology: Operative clinic—9.
JOHN McLEAN and staff. Intracapsular cataract. Glaucoma.

Friday

General surgery: Dry clinic—2.
GEORGE JULIUS HEUER and staff.
GEORGE PAPANICALAOU. Diagnosis of carcinoma from disquamated cells.
FRANK GLENN and DAN CUMMINGS GILL. Clinical results of the use of streptomycin.
SAMUEL WILSON MOORE. Follow-up studies of polyps of the large intestine.
JAMES DINGWALL. Adult tissue extract in wound healing.
ERNEST WILLIAM LAMPE. Surgical anatomy demonstration; anatomy of region involved in lobectomy and pneumonectomy.

Surgery of the bones and joints.
NELSON WARREN CORNELL—9. Operative clinic.
NELSON WARREN CORNELL—2. Dry clinic.
CHESLEY EVAN SMITH. Volkmanns' ischemic paralysis.
ARTHUR CONSOLE. Threatened Volkmanns' paralysis prevention by sympathetic block.
PRESTON WADE. Massive onlay bone grafts for ununited fractures.
BERNARD MAISEL. Table fracture of tibia; conservative treatment.
BROADMAN MARSH BOSWORTH. Fixation of acromioclavicular separation.
NELSON CORNELL. End-results; fracture of neck of femur treated with Moore nails.

Otolaryngology: Operative clinic—2.
THOMAS JOSEPH GARRICK. Surgery of the oropharynx and nasopharynx.

NEW YORK ORTHOPEDIC HOSPITAL

Monday

Operative clinics—8.

Tuesday

Presentation of postoperative patients of many years' follow-up—8.

Wednesday

Operative clinics—8.

Thursday

Grand rounds with chief surgeon at the New York Orthopedic Dispensary and Hospital, ALAN DEFOREST SMITH.

Friday

Operative clinics—8.

Monday to Friday

Out-patient clinic in the dispensary—1:30 p.m. to 4:30 p.m.

NEW YORK ORTHOPEDIC HOSPITAL FRACTURE SERVICE

Monday

SAWNIE RENARD GASTON—1:30. Problem fractures with follow-up results.

Tuesday

CLAY RAY MURRAY and staff—9. Operative clinic.
Fracture follow-up clinic—1:30.
X-ray review—4.

Wednesday

CLAY RAY MURRAY and staff—9. Operative clinic.
HARRISON LLOYD MCLAUGHLIN—1:30. Shoulder cuff injuries and recurrent shoulder dislocations with follow-up results.
BARBARA BARTLETT STIMSON—3. The low back problem with follow-up results.

Thursday

CLAY RAY MURRAY and staff—9. Operative clinic.
FREDERICK MERWIN SMITH—1:30. Complicated elbow fractures with follow-up results.
STEPHEN SYLVESTER HUDACK—3. Collagen and organic salt; combination for bone displacement.

Friday

Fracture Service Grand Rounds and Conference: Significance of blood volume studies in trauma—9.
CLAY RAY MURRAY—2:30. Rôle of sympathetic system in the surgery of trauma.

NEW YORK POST-GRADUATE HOSPITAL

Monday

Operative clinic—9. Diseases of gall bladder and bile ducts.
RUPERT FRANKLIN CARTER—11. Dry clinic. Follow-up results.
JAMES WILLIAM HINTON—2. Operative clinic.

Tuesday

Follow-up results—9.
GEORGE ANOPOL—10:30. Operative clinic.
GUSTAV AUFRIGT—10:30. Case illustrations in operative procedures in plastic surgery.
WALTER TAYLOR DANNREUTHER—2. Operative clinic: gynecology.

Wednesday

Operative clinic—9. Neurosurgery.
JOHN ARTHUR MACLEAN, JR.—9. Follow-up clinic patient illustration.
BERNARD MARRAFFINO—11. Value of immediate and delayed cholangiography in the surgical management of common duct disease. Case illustration.
Follow-up results in advanced common bile duct lesions. Case illustrations—12.
CHARLES GORDON HEYD—2. Operative clinic: General surgery.

Thursday

HERBERT WILLY MEYER and ROBERT HAYWARD KENNEDY—9. Operative and dry clinic on problems in surgery of tumors of breast, head, and neck.
HENRY HAUSMAN RITTER—2. Operative and dry clinic in traumatic surgery.
Ophthalmology—2.
RUDOLF AEBLI and HAROLD BROWN. Eye muscle operations.

Friday

CARNES WEEKS—9. Operative clinic: Surgery in hypertension.
GERALD HILLARY PRATT—10:30. Operative clinic: diseases of the vascular system.
THOMAS HENDRICK RUSSELL—2. Operative clinic: general surgery.

PRESBYTERIAN HOSPITAL

Monday

Dry clinic—2.
EDWARD LEE HOWES. Experimental production of carcinoma of stomach.
HUGH AUCHINCLOSS. Twenty year follow-up in carcinoma of the breast.
ARTHUR PURDY STOUT. The relation of chronic cystic disease to cancer of the breast.
CUSHMAN DAVIS HAAGENSEN. The problem of early diagnosis in cancer of the breast.
MAURICE LENZ. Roentgentherapy of cancer of the breast in non-operated cases and as a preoperative and postoperative treatment.
CUSHMAN DAVIS HAAGENSEN. Surgery versus radiation in cancer of the breast.

Tuesday

Dry clinic—2.
HAROLD DORTIC HARVEY. Blood volume studies in surgical patients.
OCTA CHARLES LEIGH. Physiological studies in injuries due to cold.
SIDNEY CHARLES WERNER. Use of amino acids in preoperative and postoperative nutrition.
EDWARD BALDWIN SELF. Serum versus plasma.
FENNELL PARRISH TURNER II. Use of dextran in surgical patients.
JOHN SCUDDER. Experience with human albumin.

Thursday

Dry clinic—2.
LOUIS BAUMAN. Diagnosis of pancreatic disease.
WILLIAM BARCLAY PARSONS. Surgery of tumors of the ampulla of Vater and head of pancreas.
ROBERT HARE ELLIOTT, JR. Experiences with propacil in thyroid diseases.
EDWARD LEE HOWES. Use of streptomycin and sulfomylon to prevent wound infection.
RUDOLPH NICHOLAS SCHULLINGER. Observations on acute appendicitis at Presbyterian Hospital 1916–1946.
FRANK LAMONT MELENY. Recent laboratory and clinical experiments with the new antibiotic bacitracin.

Friday

Dry clinic:
VIRGINIA KNEELAND FRANTZ. Hemostasis with absorbable gauze.
ARTHUR HENDLEY BLAKEMORE. Wiring of aneurysms.
JOHN MUNN HANFORD. Carcinoma of the tongue, follow-up results.
LAWRENCE WELLS SLOAN. Follow-up results in thyroid operations.
ROSS GOLDEN. Pathological physiology in the intestine induced by adhesions.
HENRY SAGE COOPER. Practical considerations in intestinal obstruction.

PRESBYTERIAN HOSPITAL
BABIES HOSPITAL

Friday

Members of the staff—9. Operative clinic.
Presentations of current cases—11.
Dry clinic, follow-up studies—2.
EARL C. TAYLOR. Appendicitis.
BRUCE MACLEAN HOGG. Intussusception.
LOUIS MARCEL ROUSSELOT. Banti's syndrome.
GEORGE HUMPHREYS II. Esophageal atresia.
EDWARD BALDWIN SELF. Acute osteomyelitis.

PRESBYTERIAN HOSPITAL
NEUROLOGICAL INSTITUTE

Monday

FRITZ CRAMER (time to be announced). The surgical treatment of syringopontia; the surgical treatment of the neuralgias of the facial and vagus nerves; cases of bilat-

eral parasagittal meningiomas with excision of the longitudinal sinus.

CLEMENT BUCHANAN MASSON (time to be announced). Experiences in the removal of metallic foreign bodies from the brain with the use of a Berman locator.

LESTER ADRAN MOUNT (time to be announced). Premature synostosis of the sutures of the cranial vault—a plea for early recognition and early operation. A new type of operative procedure; congenital dermal sinus, lumbosacral, thoracic, and occipital, associated with subdural abscess, meningitis or abscess of the spinal cord.

Tuesday

BYRON POLK STOOKEY (time to be announced). Dermoid tumors of the spinal cord.

JOHN EDWIN SCARFF and J. LAWRENCE POOL (time to be announced). Factors contributing to massive involuntary spasm in paraplegic patients; report on the treatment of hydrocephalus in infants.

EDWARD B. SCHLESINGER (time to be announced). Curare treatment of spastic states.

PRESBYTERIAN HOSPITAL
THE INSTITUTE OF OPHTHALMOLOGY

Monday

ALGERNON BEVERLY REESE and staff—2. Surgical procedures in glaucoma and cataract.

Tuesday

GORDON MURPHY BRUCE and staff—2. Operative clinic.

RAMON CASTROVIEJO—2. Surgery of the cornea.

Wednesday

JOHN HUGHES DUNNINGTON—2. Surgery of the extraocular muscles and intraocular operations.

ROOSEVELT HOSPITAL

Monday

Traumatic and reconstructive surgery of the hand—2.

HENRY AUGUSTUS KINGSBURY. Early treatment and management of injuries (illustrated).

R. STERLING MUELLER. Late repair and restoration of injured flexor tendons (illustrated, case presentations).

CONDUCT WALKER CUTLER, JR. Reconstructive surgery of bones of the hand (illustrated).

Tuesday

Surgical treatment of lesions of the colon—9.

HENRY WISDOM CAVE. Subtotal colectomy (operation) for ulcerative colitis. Rounds and demonstration of patients.

Surgical treatment of lesions of the colon—2.

FREDERICK HENRY AMENDOLA. Results of resection of tumors of the colon, case reports (illustrated).

HENRY WISDOM CAVE. Sequelae, complications, and late results of the surgical treatment of ulcerative colitis. (Motion pictures of technique of ileostomy and colectomy. Presentation of cases showing late results.)

WALTER WILLIAM BRANDES. Pathology of ulcerative colitis. Gross and microscopic specimens (illustrative lantern slides).

Wednesday

Cancer of the breast—9.

WILLIAM CRAWFORD WHITE. Radical mastectomy (operation). Rounds, presentation of cases and illustration of late results.

Problems of fracture therapy—2.

WILLIAM HENRY CASSEBAUM. Presentation of cases illustrating various fractures and their complications (illustrations).

MARION BECKETT HOWORTH. Management of ununited fractures of the femoral neck. Case presentations (illustrations).

JAMES EDWIN THOMPSON. Compound fracture at the knee joint (illustrated). Case reports.

Thursday

Malignant disease of the female pelvic organs—9.

WILLIAM PATRICK HEALY. Hysterectomy for cancer (operation). Presentation of cases illustrating late results in cancer of the female pelvis: discussion and demonstration of results of surgery and irradiation.

Surgery of the genitourinary tract—2.

SIMON ANTHONY BEISLER. Suprapubic prostatectomy (operation). Presentation of patients illustrating results of urological procedures.

Friday

Surgery of the stomach—9.

HOWARD A. PATTERSON. Subtotal gastrectomy (operation). Presentation of cases illustrating late results of gastric surgery, with particular reference to gastric tumors (illustrated).

WALTER WILLIAM BRANDES. Demonstration of stomach tumor specimens.

GRANT PALMER PENNOYER. The rôle of a vascular clinic in a general hospital (illustrated). Demonstration of cases.

DAVID MERRILL WEEKS. The observed effect of various drugs on intestinal activity (moving picture demonstration).

ST. LUKE'S HOSPITAL

Monday

GEORGE FRANCIS HOCH. 2. Congenital ureterectasis.

JOHN ARTHUR TAYLOR. 2:30. Testicular tumors.

CRAIG COATES. 3. Bladder tumors.

JAMES R. WAUGH. 3:30. Uteropelvic obstruction.

Otolaryngology.

WESTLEY MARSHALL HUNT. Bronchoscopic diagnosis of carcinoma of lung.

JOHN WINSTON FOWLKES. The use of penicillin in otolaryngological surgery.

Tuesday

WILLIAM FRANK MACFEE and staff—8:30. Operations.

ALEXANDER WILLIAM ADA—11. Review of surgery of stomach and duodenum.

FRED PALMER SOLLEY—11:30. Case reports—3 cases subtotal gastrectomy for massive hemorrhage.

JOHN COOPER—12. Experience with curare in prolonged operations.

MORRIS KELLOGG SMITH and GEORGE MUNRO GOODWIN—12:30. Results in surgery for toxic goiter with special reference to the use of thiouracil.

MATHER CLEVELAND—2. Hazards of external fixation in skeletal traction.

DAVID MARSH BOSWORTH—2:30. Difficult fractures. Management and end-results.

FRED THOMPSON—3. End-results in 100 trochanteric fractures of the femur.

BORIS PETROFF—4:30. Neurogenic bladder.

Wednesday

WILLIAM FRANK MACFEE and staff—8:30. Operations.

WILLIAM FRANK MACFEE—11. Carcinoma of colon at rectosigmoid junction, management.

EDWARD JOSEPH DONOVAN—11:30. Ten year study of carcinoma of colon exclusive of rectum.

PAUL CALHOUN MORTON—12. Polyps of rectum and sigmoid, management and treatment.

BENJAMIN RICE SHORE, JR.—12:30. Carcinoma of breast. Ten year follow-up report.
JOHN WINSTON FOWLKES and staff—2. Operations or ward rounds.
JOHN WINSTON FOWLKES—3. Penicillin as an adjunct to surgery in orolaryngology.
WESLEY MARSHALL HUNT—3:30. Bronchoscopic diagnosis in carcinoma of lung.

Thursday

WILLIAM FRANK MACFEE and staff—8:30. Operations.
MORRIS KELLOGG SMITH—11. Vascular surgery in war injuries.
FRANKLIN STAFFORD WEARN—11:30. Pulmonary embolism.
ALEXANDER WILLIAM ADA—12. End-results in lobectomy for bronchiectasis; case report; constrictive pericarditis (calcified), operation and end-result.
JOHN JACOB WESTERMANN, JR.—12:30. Case reports. Surgery of ulcerative colitis.

Friday

WILLIAM FRANK MACFEE and staff—8:30. Operations.
JOHN WEST—11. Inguinal hernia, study of cases and end-results.
WILLIAM BERRY—11:30. Case report, carcinoma ampulla of Vater, Operation and end-result.
HERBERT PARSONS—12. Penetrating head injuries war experience.
JOHN WEST—12:30. Case report, lymphosarcoma of small intestine with metastases to extremity.
WALTER GURNSEY FREY—2. Blood pressure variations in ophthalmological operations under local anaesthesia.
WALTER GURNSEY FREY and staff—2:30. Operations or ward rounds.

ST. VINCENT'S HOSPITAL

Tuesday through Friday

RAYMOND PETER SULLIVAN and GEORGE R. STUART and staffs—9. Surgical operations.
JOHN FRANCIS MCGRATH and staff—9. Gynecological operations.
C. G. DE GUTIERREZ-MAHONEY and staff—9. Neurosurgical operations.

Monday

Dry clinic—2. Hodgkin's disease symposium.
JOHN FRANCIS KEATING—Clinical findings.
WILLIAM WALLACE MAYER. X-ray findings and x-ray therapy.
ANTONIO ROTTINO. Pathological findings.
GLADYS CAMERON and COATA GRAND. Experimental studies.

Tuesday

Dry clinic—2. Surgical.
RAYMOND PETER SULLIVAN. Biliary tract reconstruction.
FRANK JOSEPH MCGOWAN. What civilian surgery has learned from the war.
DANIEL A. MULVIHILL. Decortication of the pleura—a new operation for non-expandable lungs.
CONSTANTINE JOSEPH MACGUIRE, JR. Pancreatitis.
BERNARD D. HANNAN. Acute cholecystitis, immediate versus delayed operation.

Wednesday

Dry clinic—2. Neurosurgical.
J. T. DANIELS. A new method for reducing extensive nerve defects.
A. GRINO. Morphology of the oligodendroglia demonstrated by a new method.
C. G. DE GUTIERREZ-MAHONEY. The treatment of phantom limb pain.
W. G. KUHN, JR. Care and rehabilitation of the paraplegic patient.

Thursday

Dry clinic—2. Surgical.
GEORGE R. STUART. Résumé of cancer studies over 4 years.
GEORGE VAN GILLUWE. Advantages of the use of compressed air in anesthesia.
EDWARD VICTOR DENNEEN. Fractures of both bones of the leg.
MAURICE CULMER O'SHEA. Fractures of the os calcis—200 cases.
T. M. HOWLEY. Carcinoma of the kidney.

Friday

Dry clinic—2. Gynecological.
JAMES PATRICK HENNESSY. Cesarian section.
ALEXANDER HUNTER SCHMITT. Multiple cesarean sections.
THOMAS EUGENE LAVELL. Ovarian neoplasma.
BERNARD JOSEPH PISANI. The program for gynecological care in the Veterans Administration.
Dry clinic—2. Otolaryngological.
JOHN MARION LORE and staff. Speech clinic, etc.

WOMAN'S HOSPITAL

Monday, Wednesday and Friday

ALBERT HERMAN ALDRIDGE, LEE MAIDMENT HURD, WILLIAM T. KENNEDY, and RALPH A. BARRETT—8:30 a.m. to 12:00 noon. Operative clinics.
Demonstrations: Treatment of neoplasms of the pelvic organs, uterine displacements, birth injuries. End-results of the procedures will be discussed.

PRELIMINARY CLINICAL PROGRAM
BROOKLYN. LONG ISLAND HOSPITALS

BROOKLYN EYE AND EAR HOSPITAL

Wednesday

Daily operative clinic.
Demonstrations by surgeons of the staff of the Brooklyn Eye and Ear Hospital.
Late results in detachment of retina.
Late results in cataract extraction.
Late results in fenestration.
Late results in dacryocystorhinostomy.

Thursday

Daily operative clinic.
Demonstrations by surgeons of the staff of the Brooklyn Eye and Ear Hospital.
Late results in facialomaxillary surgery.
Late results in laryngectomies.
Late results in glaucoma surgery.
Angioscotometry exhibit.

CUMBERLAND HOSPITAL

Monday

General surgery—2. Discussion of postoperative complications. Use of sulfanamides in acute abdomen. Preoperative and postoperative treatment in gastrointestinal surgery. Advantages of Miller Abbott tube in small bowel obstruction. Late results in the surgery of gastrointestinal malignancy with pathological demonstration.

Tuesday

Obstetrics—gynecology—9.
SAMUEL LUBIN and staff. Operations.
CHARLES H. LOUGHRAN and staff. Preoperative and postoperative care of gynecological patients.
HERBERT T. WIKLE. Procidentia, demonstration of technique for its relief, 15 year follow-up.
Late results in pelvic malignancy.
Obstetrics—gynecology—2.
Gynecological abnormalities in youth—2 case reports.
SILIK H. POLAYES. Isoimmunization with A and B factors in erythroblastosis fetalis.
LESLIE H. TISDALL. Importance of blood and blood derivatives in obstetrics.
RICHARD WALTMAN. Conservative management of abortions. Thrombophlebitis after pelvic operations.
CHARLES H. LOUGHRAN. Bacillus welchii postpartum infection—late results.

Wednesday

General surgery—9.
JOHN J. GAINEY and staff. Gastrectomy for carcinoma of the stomach with discussion of late results.
JOHN A. TIMM and staff. Ward rounds, discussion of the surgical gall bladder with late results.
HOWARD T. BLAIR. Ward rounds with discussion of surgery in large bowel malignancy. Retroperitoneal teratoma with slides.
General surgery—2.
JOSEPH I. ANTON. Arteriovenous fistula with follow-up.
SILIK H. POLAYES. Granuloma inguinale (venereum) of uterus with follow-up.
WILLIAM H. FIELD. Thoracic surgery in general hospital.
JOHN A. TIMM. Common duct atresia.
SAUL F. LIVINGSTON. Congenital esophageal atresia.
MARTIN H. LUTZ. Traumatic evisceration with follow-up.

Thursday

Fractures and other traumas—9.
LEO FASKE and staff. Pinning for femur neck fracture. Late results in fracture of femur neck.
JOSEPH I. ANTON. Traumatic abdomen.
Ward rounds with x-ray studies of various types of fractures.
Fractures and other traumas—2.
JOSEPH I. NEVINS and staff. Non-union, late results and general discussion.
Ward rounds with demonstration of apparatus in use. Gadgets and their use in fracture treatment.
FRANK TURNEY. Head injuries, late results.

Friday

Urology—9.
HOWARD T. LANGWORTHY and staff. Carcinoma of prostate. Orchiectomy for prostatic metastases—late results.
LEO S. DREXLER and staff. Carcinoma of bladder with transplantation of ureter—late results.
General surgery—2.
Thrombophlebitis after pelvic operations. Late results in thrombophlebitis and phlebothrombosis. Embolectomy for riding embolus of abdominal aorta. Convulsions during anesthesia—late results. Late results in surgical jaundice.

HOUSE OF ST. GILES THE CRIPPLE

Wednesday

JOSEPH B. L'EPISCOPO, JOSEPH GIANQUINTO, CARMELO C. VITALE. Orthopedics:
Dry clinic. Abduction traction splint for congenital dislocation of hip. Demonstration of cases. Follow-up clinic on foot reconstruction for poliomyelitic unstable feet—results following Hoke, Naughton-Dunn, Lambrinudi and astragalectomy stabilizations. Shoulder fusion for poliomyelitic flail shoulders—5 year follow-up. Demonstration of cases. Follow-up on shelf operation for congenital dislocation of hip. Presentation of cases. Recurrent club-foot. Demonstration of cases following resection of head of talus plus transplantation of anterior tibial tendon.

JEWISH HOSPITAL

Tuesday

Operative clinic—8.
LOUIS BERGER and staff.
Dry clinic—10.
LOUIS J. MORSE and JACOB S. BARB. Fascial transplants in repair of large incisional hernia.
ALFRED L. SHAPIRO. Configuration and variations of the duodenal choledochol and middle colic arteries.
LEWIS E. SCHOTTENFELD. Bacillus welchii infections of the biliary tract.
BERNARD PINES. Postoperative obstruction of the bile duct treated with vitallium tube.
HYMAN L. BERSON and PAUL W. ASCHNER. Pitfalls in the diagnosing and surgical therapy of obstructive jaundice.
BENJAMIN M. BERNSTEIN. Critical review of peptic ulcer therapy.
DAVID FARBER. Carcinoma of small bowel.

SAMUEL ACHS. Bowel perforation with Miller Abbott Tube.
LOUIS BERGER. Restroation of alimentary continuity in carcinoma of rectum.
Operative clinic—1:30.
ISADORE M. TARLOV. Plasma clot suture of peripheral nerve.
Neurosurgical dry clinic—3:30.
JOSEPH H. SIRIS and staff.

Wednesday

Operative clinics—8.
HENRY W. LOURIA and staff.
RUDOLPH NISSEN and staff.
Dry clinic—10.
IRVING M. PALLIN. Curare and endotracheal anesthesia in poor risk patients.
EDWARD HIRSCH and LEO LOEWE. Treatment of postoperative phlebothrombosis with subcutaneous heparin.
SAUL F. LIVINGSTON. Bleeding nipple.
LEW A. HOCHBERG. Paradoxical respiration; its cause, consequence, and treatment.
RUDOLPH NISSEN. Endorrhaphy as surgical procedure in the treatment of giant air cysts of the lung.
VICTOR WORONOV. The use of thiouracil in the surgical management of toxic goiter.
ARTHUR A. WEINBERG. The diagnosis and surgical treatment of thyroiditis.
FRANK TELLER. Malignant tumors of the thyroid gland—diagnosis, management and end-results.
HENRY W. LOURIA. Surgical management of goiter.
Dry clinic: Orthopedics—2.
S. A. BERNSTEIN. Operative correction of Hammer toes by resecting the middle phalanx.
MORRIS T. KOVEN. Pre-standing cases of congenital subluxation of the hip.
LOUIS S. NELSON. The conservative management of slipping of the upper femoral epiphysis.
BENJAMIN KOVEN. The use of the Austin Moore plate and its modifications in high osteotomy of the femur.
LT. COL. JOSEPH E. MILGRAM. Reconstruction Key gliding mechanisms of the upper extremity.
A. E. SOBEL and BENJAMIN KRAMER. Composition of bone in relation to blood and diet.
BERNARD S. EPSTEIN. The application of laminography to some orthopedic problems.

Thursday

Operative clinics—8.
PAUL W. ASCHNER and staff.
LEO S. SCHWARTZ and staff.
Obstetrics—gynecology—10.
E. GOLD. Pelvic drive in obstetrics.
ISIDORE DAICHMAN and WILLIAM POMERANCE. Extra peritoneal cesarean section.
HARRY J. GREENE. Phlebitis in obstetrics and gynecology.
SAMUEL A. WOLFE and IRVIN NEIGUS. Studies with gonadogen.
Dry clinic: Urology—10.
ABRAHAM D. SEGAL. Diagnosis and treatment of solitary cyst of the kidney by aspiration and injection.
LOUIS H. BARETZ. Treatment of hydrocele by sclerosing agent.
ZACHARY R. COTTLER and SAMUEL PARNASS. Periostitis and ostitis pubis complicating urological procedures.
PERRY KATZEN. Obstructive uropathy in children.
ISADORE KIMMEL and PAUL W. ASCHNER. Serum acid phosphatase in the diagnosis and management of carcinoma of the prostate.

KINGS COUNTY HOSPITAL

Monday

JOSEPH TENOPYR—10. Ward walks and demonstration of fractures.

Tuesday

GEORGE A. DIXON—9. Operative clinic including gastrectomy, etc.
JOHN F. RAYCROFT—2. Discussion of fractures of the upper humerus illustrated with lantern slides and follow-up cases.

Wednesday

JOSEPH TENOPYR—10. Demonstration of fractures and apparatus; visit to the occupational therapy department.
OTTOKAR TENOPYR. Treatment of fractured patella by complete removal of patella and suture of ligaments of knee.

KINGS COUNTY HOSPITAL—DEPARTMENT OF NEUROLOGICAL SURGERY

M. FRANK TURNEY. Glossopharyngeal neuralgia associated with cardiac arrest.
JEFFERSON BROWDER. Treatment of postherpetic pain by excision of the implicated skin.
RICHARD GRIMES. Modification of congenital spaciticity by selected dorsal root rhizotomy.
EVERETT W. CORRADINI. Diagnostic use of combined angiography and ventriculography.
ARTHUR BACON. Bilateral aneurisms—one on each common carotid artery.
JOHN P. GALLAGHER. Dorsal cordotomy for painful phantom limb.
(Time to be announced)

LONG ISLAND COLLEGE OF MEDICINE
(Kings County Hospital)

Wednesday

Dry clinic: Gastric surgery, late results. Biliary surgery, late results. Rectal surgery, late results.

Thursday

Dry clinic: Early results following sympathectomy for peripheral vascular disease. The value of open bone surgery in early restoration of the patient.

LONG ISLAND COLLEGE HOSPITAL

Tuesday

Obstetrics and gynecology—a.m.
GEORGE M. PHELAN. Surgical management of Bartholin cysts.
MERVYN V. ARMSTRONG. Addison's disease complicated by pregnancy.
WILLIAM T. DAILY. Syphilis and pregnancy.
SAMUEL A. WOLFE. Endometrial study after x-ray therapy for polyhormonal amenorrhea.
SANFORD KAMINESTER. Ectopic kidney as a complication of labor.
JOSEPH CRESI. Heart disease and pregnancy.
Urology—p.m.
FEDOR SENGER and staff. Operations, demonstrations, cases, end-results.

Wednesday

General surgery—a.m.
EMIL GOETSCH and staff. Operative program, thyroidectomy and other procedures.
EMIL GOETSCH, ARTHUR GOETSCH, ALBERT J. RITZMANN. Symposium on thyroid disorders.
GAETANO DEYOANNA. Curare in abdominal surgery.
HAROLD LAROE. Complications of Meckel's diverticulum.

Thursday

Obstetrics and gynecology—a.m.
MORRIS GLASS. Postmenopause bleeding.
ALEXANDER ROSENTHAL. Iontophoresis in the treatment of chronic pelvic infection.
ARTHUR S. MACGREGOR. Conservative management of placenta previa.
CARL TAFFEEN. Conservative treatment of absence of the vagina.
CHARLES A. WEYMULLER. Measures for the protection of newborn infants.
HARVEY B. MATTHEWS. Chronic endocervicitis, pathology, symptoms and treatment.
VINCENT P. MAZZOLA. Sodium-pentothal in major obstetric and gynecologic surgery.

Friday

Fractures and other traumas—a.m.
S. POTTER BARTLEY and HAROLD LAROE. Surgery of trauma, demonstrations, cases, end-results.
General surgery—a.m.
PAUL ANSBRO. Modern trends in anesthesia.

METHODIST HOSPITAL

Monday

Operative and dry clinic—2.
CHARLES A. ANDERSON, EINAR A. SUNDE and staff. Otolaryngology.
JOHN A. FORD. Plastic surgery.

Tuesday

Operative clinic—8.
HAROLD K. BELL, SEYMOUR G. CLARK and staff. General surgery.
HENRY P. LANGE and staff. Orthopedic surgery.
Dry clinic—2.
HENRY F. GRAHAM, moderator. Symposium on gastric surgery.

Wednesday

Operative clinic—8.
PIERRE A. RENAUD, JOHN A. TIMM and staff. General surgery.
HOWARD T. LANGWORTHY and staff. Genitourinary surgery.
Dry clinic—2.
ARTHUR H. BOGART, moderator. General surgery and specialties.

Thursday

Operative clinic: Obstetrics and gynecology—8.
GEORGE H. DAVIS, HARRY W. MAYES, HENRY P. HAGSTROM, HENRY S. ACKEN, STANLEY C. HALL and staff.

Friday

Tumor clinic (late results)—9.
Hospital staff. PIERRE A. RENAUD, moderator.

ST. JOHN'S HOSPITAL

Monday

General surgery—8.
Operative clinic. Ward rounds.
Plastic surgery—8.
Operative clinic.. End-results in cleft palate. Demonstration of cases.

Tuesday

Gastrointestinal surgery—8.
Operative clinic. Ward rounds on gastro-intestinal cases. Conference in x-ray department. Conference in tissue laboratory. End-results in duodenal ulcer.
Thoracic surgery—8.
Operative clinic. Preoperative and postoperative care. Ward rounds—dry clinic. Discussion: Surgical treatment of bronchiectasis.

Wednesday

Goiter surgery—8.
Operative clinic. Preoperative and postoperative care. The abuse of thiouracil in hyperthyroidism. Ward rounds. End-results in toxic goiter.
Urology—8.
Operative clinic. End-results. Heminephrectomy for horse shoe kidney. Cancer of ureter—end-results. End-results in cancer of the bladder.

Thursday

Fractures and other traumas—8.
Operative clinic. Visit to fracture clinic. X-ray demonstrations of interesting fractures. End-results in fractures of the femur ward rounds.
Gynecology—8.
Operative clinic. Demonstration of gross specimens of ovarian tumors. End-results in tumors of the ovary.

Friday

Surgery of the hand—8.
Operative clinic. Visit to the hand clinic. Motion pictures of operations for lesions of the hand—end-results. Round table discussion.
Ear, nose and throat surgery—8.
Operative clinic. Endoscopic procedures. Conference in x-ray department. General discussion.